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SOME OBSERVATIONS ON PEPTIC ULCERATION: WITH SPECIAL REFERENCE TO ITS MEDICAL MANAGEMENT.[1]

By C. F. PITCHER, M.B., B.S. (Adelaide),
Adelaide.

WHEN asked by the Medical Secretary to read a paper before the Society, I felt sensible of the honour conferred on a general practitioner who does mostly lodge work and at the moment it appeared difficult for me to find a subject which would be of interest and, I dared to hope, informative to its members. During the last six years I have been actively engaged in practice at Hindmarsh and during that period have seen with my partner, Dr. Bonnin, thirty-eight patients with peptic ulceration. As this represents some of the grain garnered from the much too much chaff of a busy lodge practice, I decided on a paper on peptic ulceration. Another reason for deciding on peptic ulceration is the divergence of opinion between the surgeon and physician in regard to treatment. This I hope will be provocative of criticism which will be more instructive than the paper.

I intend to survey lightly the whole field of peptic ulceration, as after all it is only a matter of anatomical geography and the ætiology is probably identical.

Incidence.

Owing to the improved diagnostic technique and more scientific investigation, peptic ulcer has come to be recognized as a fairly common disease. Fifteen years ago when we were students and house surgeons at the Adelaide Hospital, peptic ulceration did not loom large in our eyes. Is this increase in the incidence all due to our improved methods of investigation or are the other factors to be considered? Generally speaking, duodenal ulcers are more common in men and gastric ulcers in women.

Ætiology.

The cause of peptic ulceration has not been established satisfactorily. One knows that there are two essential factors: (i.) Causes which act by damaging the mucosa and (ii.) digestive action of gastric juice.

[1] Read at a meeting of the South Australian Branch of the British Medical Association on March 25, 1926.

There are two sets of causes which are held to act by injuring the mucosa. Virchow maintained that ulceration was due to vascular blocking, by arterial embolism or by venous thrombosis. He deduced this by the peculiar funnel-shaped appearance of some of the acute ulcers. The more recent theory of the bacterial origin of peptic ulcers has received universal support in America, but in England and on the continent there is no such unanimity.

In 1857 Labat produced experimentally acute ulcers of the stomach by the intravenous injection of pus as part of a general pyæmia. This strongly suggested that acute ulcers of the stomach and duodenum could be produced by embolic bacterial invasion.

Rosenow in 1913, using streptococci of low virulence as an intravenous injection, produced ulcers of stomach and duodenum. The streptococci were obtained from various sources, such as alveolar abscesses and infected tonsils and he concluded that the ulceration is due to localized infection and secondary digestion. In 1915 Rosenow demonstrated streptococci in the bases of gastric and duodenal ulcers. This was confirmed in England by Bolton, Stewart and several others. In 1921 he brought forward his "electric localization phenomenon." According to this streptococci recovered from an appendiceal abscess will reproduce similar lesions if injected into animals. Considerable doubt is thrown on this in England, as several competent authorities have failed to reproduce his results. He also demonstrated that streptococci, recovered from the lesion in cases of appendicitis, cholecystitis, endocarditis, tonsillar and apical dental abscess, have a special affinity for the gastric mucous membrane.

A chronic ulcer is only an acute ulcer which has failed to heal and there are several facts which contribute to its delay. The chief of these are: (i.) Corrosive action of the gastric juice and (ii.) neuro-muscular instability.

Morbid Anatomy.

It is significant that gastric and duodenal ulcer occur only where the tissue is exposed to the action of the gastric juice. For the most part gastric ulcers occur along the lesser curvature and on the posterior wall close up to the pylorus. Duodenal ulcers occur chiefly within 3·75 centimetres (one and a half inches) of the pylorus, that is within that portion of the duodenum which retains the gastric contents longer than any other or as Mayo remarks, "only within this neutralizing field can an ulcer occur." Ulcers are generally single, although Moynihan states that in 12% of cases there are more than one. They vary in size and shape and from their healing two definite pathological conditions result: (i.) Stenosis of the pyloric outlet, (ii.) hour glass stomach.

Symptoms.

Ulceration may exist without any symptoms. I have had one experience.

For six months I treated a patient for an injury to his knee caused by his having fallen a distance of nine metres (thirty feet) into a hole. On the evening that he came to be put off the funds of the lodge, he asked for a bottle of indigestion mixture as he had some discomfort after the three previous meals. At five o'clock the next morning he sent for me and he was found to be suffering from an acutely inflamed abdominal cavity due to a perforated duodenal ulcer for which he was operated on immediately in the Adelaide Hospital. Unfortunately, however, he developed an empyema and died six months later of an embolic cerebral abscess.

This man was a lodge patient and he had never complained of any trouble until that night. It is quite possible that he was suffering from one of these acute ulcers of twenty-four hours' standing. He recovered completely from his operation, but developed an empyema and died.

Pain or discomfort is the most prominent symptom of ulceration. It varies in character, site and periodicity. In gastric ulcer pain comes on earlier in the digestive cycle. It is variously described as gnawing, burning and epigastric in situation. Usually the pain in gastric ulcer occurs immediately after the ingestion of food and is relieved by vomiting and by alkalis. The nearer the ulcer is situated to the pylorus, the shorter is the period of freedom from distress after the taking of food. I have never satisfied myself about the pain and tenderness to the right of the tenth dorsal vertebra which is so often described. In this series it was conspicuous by its absence.

In duodenal ulcer the classical Moynihan syndrome is seen. This consists in epigastric pain slightly to the right of the middle line, coming on two to three hours after the taking of food and relieved by ingestion of food and alkalis. By careful history-taking this syndrome may be elicited in nearly every case.

The cause of pain in duodenal ulcer is still obscure. There are two hypotheses.

The acid hypothesis presupposes a corrosive action of the acid on the ulcer. This view is rather discounted by the Rehfuss fractional meal which discloses that the pain does not occur at the height of the acid curve. It is seen by the fluoroscope to take place at the height of motor activity of the stomach.

The tension hypothesis is now the more popular. According to this the ulcer irritates the neuromuscular structures and the pylorus fails to relax in a normal way, this results in a condition of spasm. For this phenomenon of failure of the pylorus to relax Hurst has recently coined the new word achalasia.

Vomiting had not been a symptom of any importance in our series. Of course it is prominent in those conditions which result from healing of an ulcer, conditions such as pyloric stenosis and hour glass stomach.

Tenderness is not a constant symptom. A history is often obtained of relief of pain by pressure on the epigastrium.

Melæna and hæmatemesis were fairly constant in our series and it is remarkable what a number of ulcers apparently begin their career with hæmatemesis.

Diagnosis.

Three things are essential to the making of a correct diagnosis:

(i.) The taking of an accurate history, the patient being impressed with the necessity of timing accurately his meals and distress.

(ii.) Analysis of the stomach contents by means of an Ewald one hour test meal and a Rehfuss fractional test meal. In this way an acid curve may be obtained.

(iii.) X ray examination. Dr. McCoy will go fully into this part of the subject.

There is one condition for which ulceration may be commonly mistaken, and that is disease of the gall bladder and *vice versa*. It is now quite possible by means of the intravenous injection of a dye and the use of X rays to outline the gall bladder and this will probably eliminate one of the many pitfalls of the upper part of the abdomen. In regard to malignant disease we were taught that it was fairly common for a gastric ulcer to become malignant, but now English authorities do not hold this view. Of course, it is common knowledge that malignant change is never superimposed on a duodenal ulcer. In malignant disease neither the results of X ray examination nor the findings on analysis of the stomach contents offer infallible evidence and it is the duty of the physician, if he is in doubt, to call in a surgeon.

Four years ago I sent an elderly woman to the hospital. She had a visible and palpable swelling in the epigastrium. She was detained six weeks. Analysis of the stomach contents revealed normal hydrochloric acid and the radiologist reported a spasmodic condition of the stomach, namely a functional rather than a structural condition. About a month later my partner opened her and found an unremovable mass involving the lower third of the stomach.

Treatment.

I think that there are definite indications which bring the treatment of peptic ulceration into the domain of the surgeon. I will enumerate the following:

(i.) Perforation into the peritoneal cavity which brooks of no delay.

(ii.) Hour glass stomach.

(iii.) Pyloric obstruction which has resisted efficient medical treatment.

(iv.) Any suspicion of malignant disease.

I consider that patients with severe hæmorrhage should be treated by medical means, as operation on individuals with a low percentage of hæmoglobin constitutes a grave risk. Blood transfusion is of great value in these cases.

In all cases foci of infection should be most diligently sought. Teeth, tonsils, nasal sinuses and kidneys should be most thoroughly examined. Moreover, the surgeon if he attacks the condition, should examine the gall bladder and appendix for any sign of infection. I believe that Moynihan, Mayo and Sherren always examine these organs when operating on a patient with peptic ulcer.

In the medical management of these cases I am guided chiefly by Hurst and Bolton in a modified Sippy *régime*. The principle involved is the restoration of the normal digestive cycle. We may do this (i.) by reducing the amount of gastric juice secreted and (ii.) by reducing the motor activity of the stomach. By the latter we relax the pylorus and obtain an efficient neutralization by the regurgitation of alkaline contents of the duodenum.

The patient is put to bed for from three to six weeks. Rest is essential as it reduces the expenditure of energy. Less food is required and this gives the stomach a certain amount of physiological rest. I then order from one hundred and twenty to two hundred and ten cubic centimetres (four to seven fluid ounces) of whole milk to be given every three hours from 7 a.m. to 10 p.m. One and a half hours after each feed I order a powder of 1·2 grammes (twenty grains) of heavy calcified magnesia. This is the time when the acidity is at its height. My reason for using calcified magnesia as a neutralizing powder is that it has four times the effect of carbonate of soda and is not so liable to set up alkalosis.

Izod Bennett has shown that carbonate of soda tends to excite the gastric mucosa to increased secretion which more than counterbalances its neutralizing effects.

After three or four days of this diet I allow three feeds of about sixty cubic centimetres (two fluid ounces) of any well cooked cereal to take the place of three milk feeds. At the end of the first week I give two or three soft eggs with cereal and milk which I continue for a week. Then I substitute cream soups, custards and vegetable *purées* in order to build up three substantial meals a day with the milk. At the end of a month patients are put on to three meals a day, the meals consisting of eggs, cereal and vegetable *purées*. Bread and butter and seedless jams are allowed. Milk is given at 11 a.m. and at 3 p.m. After another fortnight the majority of patients return to their employment and take with them a "Thermos" flask of milk and neutralizing powder. Meat is forbidden for three months and then given in the form of fresh minced mutton, lamb, chicken and rabbit.

Bolton and Izod Bennett have shown that meat excites a maximal amount of gastric juice and is very hard to disintegrate.

This is the chief objection to the Lenhartz diet which aims at light protein feeding. Tobacco is withheld on the ground that it increases motor and secretory activity of the stomach. The patients are kept under strict surveillance for at least six months and they are warned to be very careful of their diet for at least two years. Alkalis are administered for at least six months. Under this *régime* I claim to have been fairly successful in relieving the distress of my patients and as the history of some of them dates back five or six years without any recurrence, I have some justification for continuing it.

Originally Sippy gave hourly feeds with alkalis between each. He used a ninety cubic centimetre (three fluid ounce) mixture of cream and milk in equal parts. By this he claimed complete neutralization, but it is probably quite unnecessary as it may actually stop digestion. He used large doses

of alternating powders, one constipating and the other laxative in effect. I abandoned this method, as most of my patients found it irksome and rather nauseating. Another objection is the risk of alkalosis, a condition of intoxication whose symptoms are similar to those of tetany. The symptoms consist in twitching of muscles with convulsions, nausea, vomiting, hiccough, dryness and redness of the skin, œdema of the extremities, drowsiness which proceeds to coma, and sometimes anæmia which may be fatal. I have never seen a case, but I believe several have been reported in America after the strict Sippy *régime*.

All my patients with hæmorrhage are put under the influence of morphine and kept so for forty-eight hours. Nothing is given by the mouth for two days save ice to assuage the thirst. Then they follow the above *régime*.

This may not meet with the approval of some who believe in prolonged oral starvation for hæmetemesis. I have not seen any bad results from the early feeding of these patients. In regard to the olive oil treatment adopted by Cohnheim on the supposition that fat inhibits the secretion of gastric juice, this has had a fair trial in my series. But most of the patients complained of eructations and nausea and so I abandoned it. I certainly impress on all my patients that the price of cure is "eternal vigilance" over the diet.

Case Histories.

I wish now to report to you four cases of peptic ulceration which I must describe as both surgical and medical failures.

CASE I.—The patient, P.P., is now fifty-seven years of age. He is a non-smoker, a teetotaller and wears artificial dentures. He was operated on by Dr. Cudmore in June, 1918. Posterior gastro-enterostomy was performed for duodenal ulcer of twenty-five years' standing with the classical Moynihan's syndrome. Previous to this operation he had both melæna and hæmatemesis on two occasions in 1902 and 1903. He had complete relief from all symptoms for four months after the operation and then experienced occasional discomfort. He was first seen by me no June 25, 1920. At this time he was suffering from syncope and all the signs of internal hæmorrhage, with pain in the epigastric region. After two or three hours he had a large hæmetemesis of about two litres with some food material. Next morning examination of the stools revealed melæna. He was kept in bed for six weeks and was given Sippy diet and alkalis. He did not improve. The pain was of varying periodicity and was not relieved by the taking of food. He was submitted to operation on November 8, 1920.

At the operation it was found that the duodenal ulcer had healed, but that a large shallow ulcer was present on the lesser curvature. The stoma of the gastro-enterostomy was quite normal and had not contracted. The duodenum was divided and closed by a purse string suture. The whole of the ulcerated area was excised by partial gastrectomy. Laboratory examination of the ulcerated surface revealed no evidence of malignant disease. For two years the patient had complete freedom from all pain with the aid of a modified Sippy diet. In December, 1922, he had a severe hæmorrhage with a recurrence of pain. The site of pain during this attack was entirely different and was referred to the left iliac region. Epigastric discomfort was conspicuous by its absence. The patient was quite definite about the alteration of the site of the pain and in my opinion he had a gastro-jejunal ulceration. He was treated for three months in bed on the Sippy diet. He improved and remained free from pain and hæmorrhage until February, 1924. He then had a severe attack of melæna with pain, referred again to the left iliac region. Blood examination revealed a hæmoglobin percentage of 27 and a typical picture of secondary anæmia. After two months in bed on a strict Sippy diet he improved and has remained well until the present time. He has of course a very strict dietary and takes alkalis regularly. After the last attack I ordered parathyreoid extract (Parke, Davis and Company) in doses of 0·006 gramme (one-tenth of a grain) to be taken for six months as recommended by Hurst. Although this endocrine therapy is criticized and pronounced to be both irrational and unscientific by Vincent and Collip, yet this man declares that he has never been better in health than at the present time for the last thirty years.

There are several interesting features in this case. In the first place the severe hæmorrhages have extended over a period of twenty-five years. Secondly there was the formation of an ulcer on the lesser curvature with a perfectly acting stoma of a gastro-enterostomy. Moynihan states that in 12½% of cases both duodenal and gastric ulcers are present. This rather disposes of the view that gastro-enterostomy is a cure for peptic ulceration, as some of our surgical friends would have us believe. It certainly suggests to me that until the ætiology of peptic ulceration is more definitely settled, we cannot hold out for these people anything more than temporary relief. In the third place the site of the pain altered after the second operation. Epigastric pain and discomfort were his constant companions for twenty-five long years, then he was suddenly seized with a pain varying in character and situation. In my opinion the alteration of the site of pain was due to a gastro-jejunal ulcer and with this view Dr. Cudmore concurred.

CASE II.—A.P., a female, aged forty-six years, was first seen by me in July, 1921. She complained of a "lump in the stomach," indigestion and loss of weight. The symptoms had been gradually getting worse for the previous fifteen years and the lump had been noticed for the previous two years. She had been operated on by the late Dr. Poulton twenty-one years previously in the North Adelaide Private Hospital for a perforated gastric ulcer. On examining her I found her to be a very miserable woman. Her teeth were decayed and septic and pyorrhœa was present. A scar was present in the middle line of the abdomen above the umbilicus and a hard mass, moving extensively with respiration, was palpable. The mass was tender on pressure. X ray examination revealed hour glass constriction of the stomach. She was ordered to have all her teeth extracted, but refused. She was operated on by Dr. Cudmore on August 16, 1921. Hour glass constriction was discovered at operation and gastro-enterostomy was performed after the dense perigastric adhesions had been broken down. She made an uninterrupted recovery. The teeth were extracted under ether anæsthesia two months later. She did very well for three years and put on about twenty-five kilograms (fifty-six pounds) in weight. In January, 1925, she complained of pain and discomfort after food with no regular periodicity. She said that the pain was the same as she had twenty-five years previously and was not like the pain which she experienced before the operation. She told me that she had another "ulcer." I thought that the constriction had become narrow again and that the perigastric adhesions had reformed, as her pain was accentuated by deep respiration. She was again examined by X rays and a report was received that the hour glass constriction had again become narrow. She was operated on by Dr. Cudmore on March 11, 1925. The stomach was opened and the constriction was found to admit three fingers easily. Just above the hour glass constriction, however, a deep ulcer about the size of a sixpenny piece was found on the posterior wall of the stomach close to the lesser curvature.

The ulcer had very ragged walls and its base was formed by the muscular coat. The ulcer was excised by a wedge shaped incision and posterior gastro-enterostomy was performed. Convalescence was of necessity slow after this rather severe operation. She reported to me about six weeks later that she had no discomfort, but felt very weak. She is at present convalescing at Victor Harbour and I believe that she is free from all symptoms and is putting on weight.

One of the interesting points about this case is that the patient diagnosed her condition accurately. She was certain that she had another ulcer. She was quite convinced that the pain was exactly the same as she had experienced twenty-five years previously and that it was in no way similar to the pain from which she suffered for fifteen years, while the hour glass contraction was slowly forming. The patient had a very foul mouth and all her teeth were extracted two years before the second ulcer formed. No other septic focus was discovered. Since the second operation she has been on a Sippy diet with alkalis and I intend to keep her on this for two years. It is quite possible that the patient did not pay sufficient attention to her diet and that this may have accounted partly for her second ulceration.

I wish to thank Dr. Cudmore who was associated surgically with me in the treatment of these patients for his help and for access to his surgical notes.

My next two cases are of interest in that the patients are father and son.

CASE III.—The patient, J.W., aged fifty-four years, was a moderately heavy smoker and gave no history of alcoholism. For fourteen years he suffered from pain in the abdomen relieved by food, alkalis and vomiting. He was first seen by me in April, 1921, while he was suffering from a severe hæmorrhage (melæna and hæmetemesis). He was given a Sippy diet and was kept at rest in bed for six weeks. He was well for two years and eight months and experienced no discomfort whatever. He then had another hæmorrhage and was put on Sippy diet and made to rest in bed. He had freedom from all symptoms until August, 1924, when he had another hæmorrhage. He was admitted to the Adelaide Hospital and was submitted to operation on August 4, 1924. According to the notes obtained from the Adelaide Hospital a small indurated ulcer about the size of a sixpenny piece was found on the posterior wall of the duodenum about 1·25 centimetres (half an inch) beyond the pylorus. Posterior gastro-enterostomy was performed. He obtained immediate relief of all his symptoms. He says that he was told that he might eat anything and he did so after his discharge. He paid no attention to his diet and did not take alkalis. Twelve months afterwards I was called to see him again at about three o'clock in the morning, when he was suffering from another severe hæmorrhage and I ordered his removal to hospital on August 1, 1925. He was in Adelaide Hospital for six weeks on a Sippy diet and alkalis and also suffered from pneumonia. Since then he has been on a strict diet and has taken alkalis regularly. He has returned to work with complete freedom from discomfort after food.

This man was discharged from the hospital about three weeks after gastro-enterostomy and was told by a nurse that he might eat anything. In my opinion these peptic ulcer patients cannot eat anything and I maintain that they should be kept on a Sippy diet for six weeks after their surgical treatment and on a modified diet for another year.

CASE IV.—J.W., aged twenty-seven, was a non-smoker and a teetotaller. He was first seen by me in November, 1922. He gave a history of pain immediately after food of two months' duration. He then suffered from hæmetemesis and melæna. He was sent to the Adelaide Hospital, was put on Sippy diet and alkalis for three weeks and was discharged free from all symptoms. He was moderately careful with his diet until July, 1923, when he had another severe attack of pain and melæna. He was admitted to the Adelaide Hospital and operated upon on August 10, 1923. According to the Adelaide Hospital notes a small ulcer was found near the pylorus on the lesser curvature of the stomach. Posterior gastro-enterostomy was performed. He was discharged on September 30, 1923, and was told by the nurse in charge of the ward that he might eat anything. Three months later he had melæna for seven days. He was then put on strict Sippy treatment. For two years he was free from pain and hæmorrhage until he got careless with his diet and had another hæmorrhage in October, 1925, and still another on November 15, 1925. He was put to bed for six weeks and still has a very modified diet. At present he is convalescing with freedom from pain.

In all these cases both medical and surgical treatment have apparently failed. It is apparent that these patients will have to be very careful of their intake. They are practically condemned to a "pappy" diet with continuous taking of alkalis.

Conclusions.

I hold that all patients with peptic ulcers in their early stages should be treated medically. I believe that even Moynihan who calls himself the physician led by force of circumstances to practise surgery, has said that it is at least arguable that the necessity for surgical relief in many cases is due to a too perfunctory trial of medical treatment. He rightly blames the physician. How many of us have had patients with a history of indigestion for months; we have comforted them and sent them off with a bottle of bismuth and soda or else with an acid pepsin mixture, as our fancy dictates. No wonder that they wander from doctor to doctor until someone decides to give them a test meal and treat them rationally. Of course, in a lodge practice it is very difficult to get these tests done and we look forward to the time when we shall be able to send these patients for an analysis of the stomach contents or for an X ray examination at some public institution from which they will be returned to their own doctor. Nowadays if they are sent to an out-patients' department, they are detained for a few weeks. The general practitioner only needs to have the tests made, he can treat the patient quite as efficiently as those on the staff of a public hospital.

Another point which I would like to bring forward tonight, is the manner in which these patients are discharged, when they have been treated either by medical or surgical means. They should report to their own doctor who will no doubt order a modified Sippy treatment, or else they should be rationally dieted by the honoraries. I have seen fourteen patients in the last three years discharged from the Adelaide Hospital after treatment for peptic ulceration and not a single one was told what he should eat. On questioning them I was told by the majority that the sister or nurse told them that they might eat anything. I wish to be emphatic on the point that neither the surgeon with his "physiological" operation nor the physician with his Sippy treatment have cured these patients.

There is no such thing as a quick cure in gastric ulceration. They must be ever watchful of their intake, the slightest deviation brings untold misery to these chronic sufferers.

Now a word to the surgeons who maintain that this method is the only treatment for peptic ulceration. Remember that in spite of gastro-jejunostomies, partial gastrectomies and innumerable other mutilations which may be the fashion of the day, your relief is only temporary; so put the patient after you have operated on him on a Sippy diet for six months, diet him carefully for another year and probably he will not be seen on your doorstep again.

Finally it behoves both physician and surgeon to be more tolerant and less dogmatic in the treatment, until either the pathologist or biochemist sheds some light on the obscure ætiology of this not uncommon disease.

THE X RAY DIAGNOSIS OF DUODENAL ULCER.[1]

By H. A. McCoy, M.B., Ch.M. (Sydney), D.M.R.E. (Cantab.), *Adelaide.*

Introduction.

BEFORE considering our present knowledge of the radiological recognition of duodenal lesions, it should be interesting to review the development of one technique which has rendered possible such recognition—a technique which has been employed and elaborated by Scott and Vilvandré in London, Barclay in Manchester and Carman at the Mayo Clinic in America.

Early in this century substances opaque to X rays were used for the purpose of studying the contour of the hollow viscera, the opaque materials after ingestion acting on the lumina of the hollow viscera as casts which could be radiographed. Abnormalities in the contour of a viscus which had been completely filled, were recognized in this way.

It was possible to study satisfactorily the size and shape of the stomach by these means, for when filled by ingestion the pylorus, in performing its ordinary function, caused a retention of the opaque substance in the stomach for a sufficient period for a radiograph to be taken. It was recognized, however, that circumstances rendered a similar study of the duodenum more difficult. The rapidity of the passage of opaque material through the duodenum and the absence of any sphincter control to its outlet, coupled with the intermittent character of the passage of the material through the pylorus are factors which obviate the probability of the lumen of the duodenum being distended at any time in the natural course of events. Hence it was decided that radiographs revealed as a rule an incompletely filled duodenum.

The first method introduced to combat this difficulty was originated by Gregory Cole, of New York, who employed serial radiography of the duodenum. Exposures were made consecutively at short intervals and a comparison of the shadows obtained was made for the purpose of estimating the size and shape of at any rate the first stage of the duodenum or duodenal cap.

In this way it was found that the normally filled duodenal cap appeared on the radiograph as a blunt cone and any departure from this general appearance reproduced on all of the radiographs, indicated the presence of an abnormal cap.

Besides being cumbersome and expensive, this method proved itself to be unreliable in many instances and a further development became imminent. Fluoroscopy, then, was employed for the purpose of visualizing the duodenal cap. From the outset it had much to recommend it, for the filling and emptying of the cap could be seen as a complete process on many consecutive occasions, as against the reproduction of shadows representing the duodenal cap at irregular periods of this process.

Appearances of a Normal Duodenal Cap.

As an introduction to the technique to be described a clear idea of the shape of a normal cap is essential. It appears, when completely filled with opaque material, as a bluntly conical shadow with smooth convex sides and a straight base, the centre of which overlies the pylorus.

The long axis of the cone occupies various degrees of obliquity, regulated by the type of stomach present. In the average orthotonic J-shaped stomach the apex lies between the positions of ten and eleven o'clock measured from the pylorus. In a hypertonic stomach of the "steer horn" variety the axis may be transverse and is sometimes directed posteriorly so that in the postero-anterior direction the cap appears small and rounded.

All degrees of size of the cap and obliquity of its long axis are met with in the normal subject and such variations must be taken into consideration in discussions on abnormalities.

The truncated apex of the cap owes its appearance to the turning posteriorly of the terminal portion of the first stage of the duodenum which is seen in the lateral view merging into the descending portion or second stage.

The clear cut sides of the cap are in marked contrast to the feathery appearance of the shadow of the second and third stages of the duodenum. The difference is due to the presence in the latter portions of *valvulæ conniventes* which encroach on the lumen and displace the opaque material. The absence of these structures in the first stage thus accounts for the variation in appearances.

Technique.

The patient stands during the first part of the examination and is examined with the screen in the lying posture only as a rule to confirm the findings noted when he is in the standing posture.

The opaque salt (usually barium sulphate) made up in the form of an emulsion, suitably flavoured,

[1] Read at a meeting of the South Australian Branch of the British Medical Association on March 25, 1926.

ILLUSTRATIONS TO DR. J. F. MACKEDDIE'S ARTICLE.

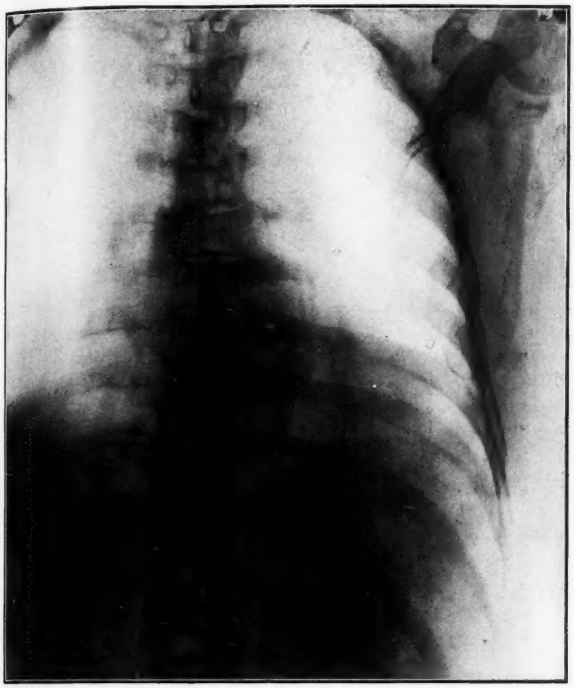

FIGURE I.

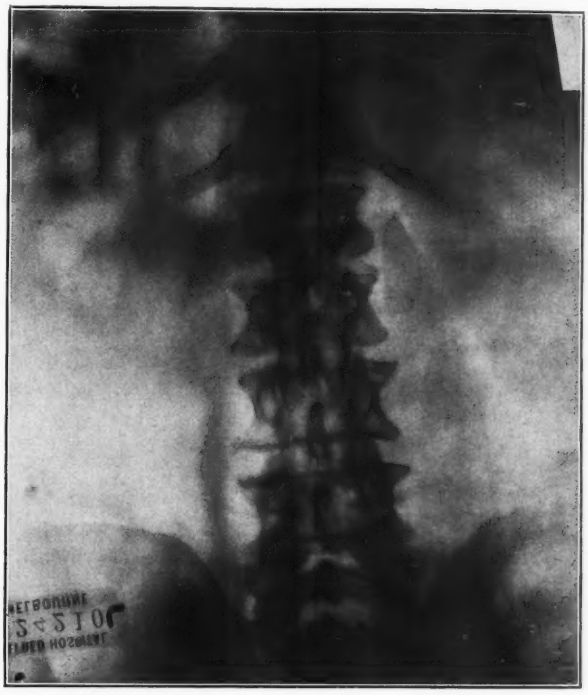

FIGURE II.

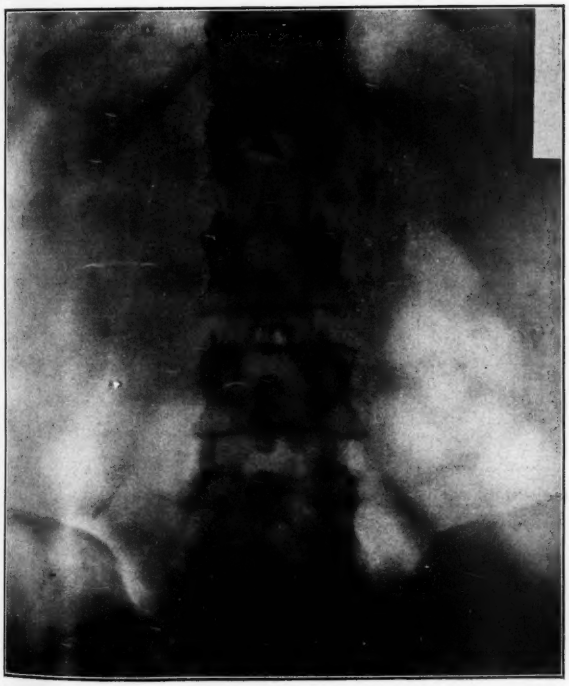

FIGURE III.

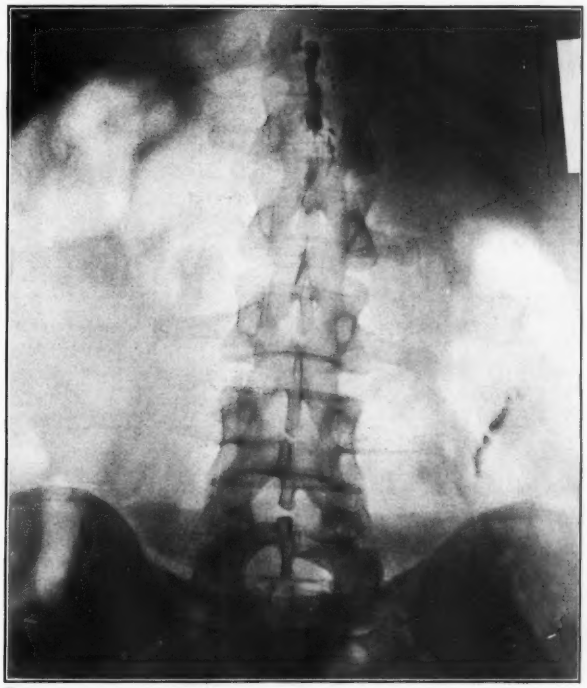

FIGURE IV.

ILLUSTRATIONS TO DR. J. F. MACKEDDIE'S ARTICLE.

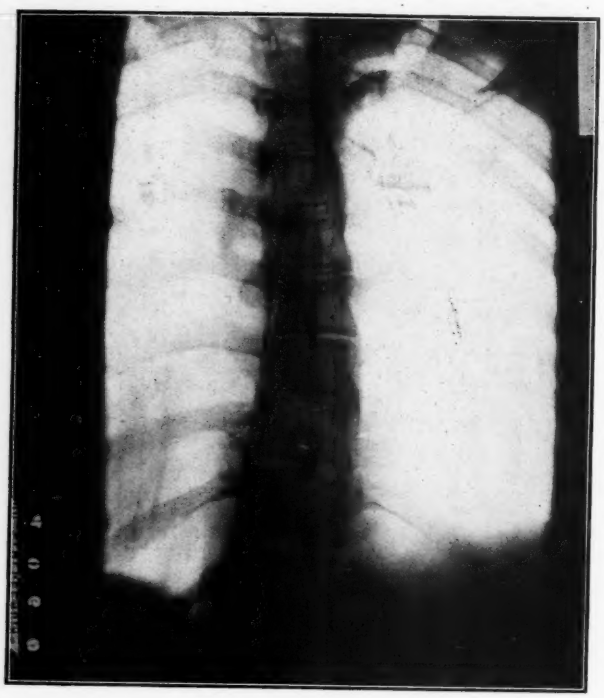

FIGURE V.

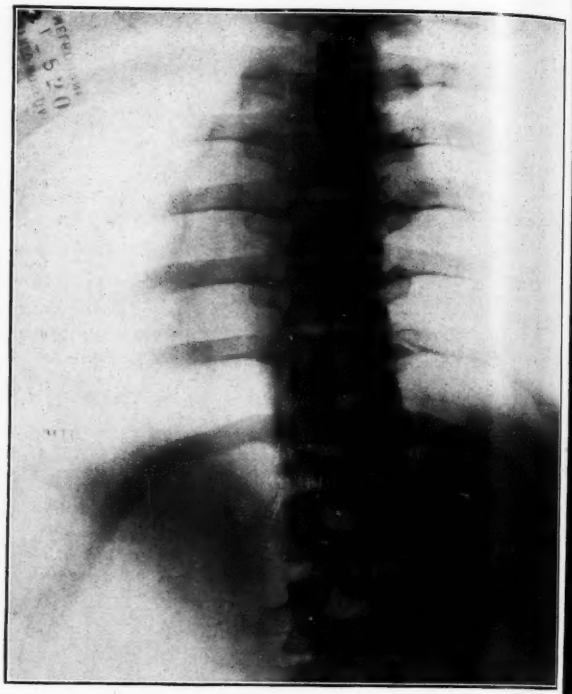

FIGURE VI.

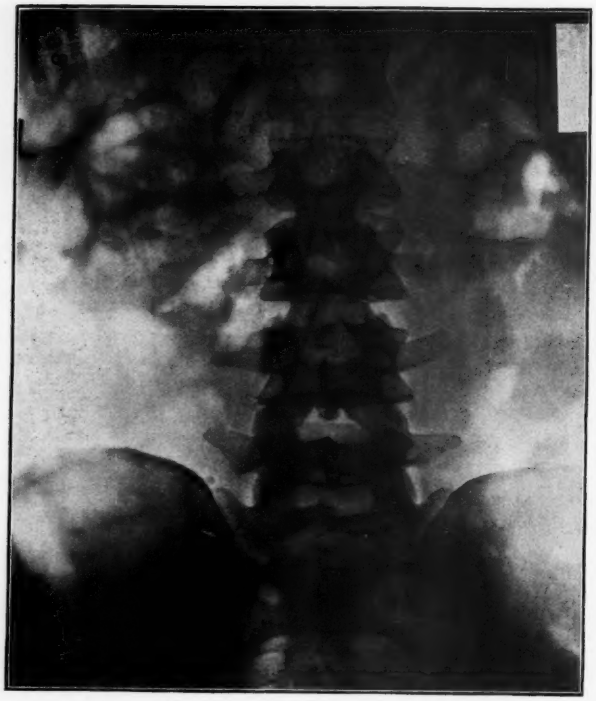

FIGURE VII.

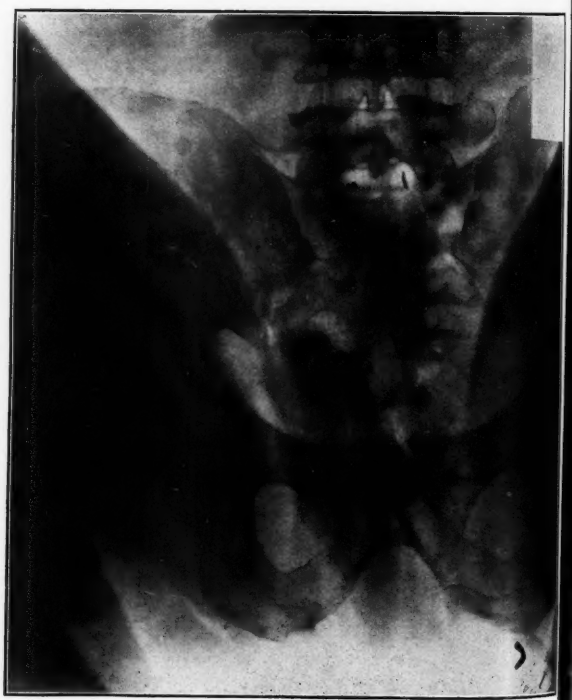

FIGURE VIII.

is ingested and its progress is viewed on the fluorescent screen. The viscera examined consecutively are thus the œsophagus, stomach and duodenum. When the shadow reaches the cardiac orifice of the stomach palpation begins and a thorough examination of that organ is made, details of which do not concern this paper. This examination may frequently be completed before any of the meal enters the duodenum.

Watching carefully the peristaltic waves of the stomach, the examiner takes advantage of each wave as it reaches the pylorus to augment the peristaltic action by gentle manual pressure directed from the pyloric antrum towards the pylorus. In this way peristalsis is stimulated and a quantity of meal greater than the average normal is forced into the duodenum with each wave.

By this manipulation the cap is filled to its normal limits for a sufficiently long period to enable the examiner to note its size and shape. Rotation of the patient to left and right allows of a study of each vertical radial section of the cap. This process is repeated many times until the cap has been seen fully distended on several occasions.

Frequently and especially in cases of acute duodenal ulcer hyperperistalsis of the duodenum prevents adequate distension of the cap after this manipulation. Firm pressure with the thumb of the left hand over the descending portion is found to obstruct its lumen sufficiently to allow of complete filling in spite of such irritability.

Having examined the outline it is now desirable to search for abnormalities in the walls of the cap. Its contents are expressed by manual pressure and in the process the opaque material is massaged into the walls or to use Scott's expression "the walls of the cap are whitewashed." By this manipulation opaque material is forced into any recess which may be present in the mucosa, as for example into an ulcer crater. The gumming together of the edges of a crater by exudate or the plugging of its lumen by a fragment of food substance transparent to X rays may easily prevent the entrance of the barium salt in the ordinary course of events. Efficient massage, however, as a rule dislodges the transparent particle or breaks down the temporary cohesion.

If then an ulcer crater be present, it appears as a dense black spot in a region which has been manually cleared of opaque substance. Such an appearance, observed unmistakably on repeating the manipulation many times, is diagnostic of a duodenal ulcer.

Although in this technique the examination of the cap is the most important factor, valuable information is often to be gained from the condition of the stomach. Such information may supply indirect evidence of a duodenal lesion. Hence the type of stomach, its peristalsis and motility are carefully noted.

Of these factors peristalsis is the most important and in the normal subject it is customary to see two well developed waves of peristalsis passing through the body of the stomach at one time. The presence of more than three fully formed waves indicates hyperperistalsis. Carman's description of this phenomenon is worthy of note:

> It [hyperperistalsis] varies from a slight increase of wave depth and frequency to a tempestuous energy of contraction. It is most exaggerated in the obstructive cases, but it occurs also when there is no obstruction. A characteristic feature is the regular succession and symmetrical correspondence of the waves on both curvatures which are equally indented. Three or four pairs are seen in progress at once, whereas only one or two pairs are seen normally.

Radiological Signs of Duodenal Ulcer.

The radiological signs of duodenal ulcer are direct and indirect. The direct signs are two in number:

(i.) The presence of a crater.
(ii.) Deformity of the cap.

The indirect signs are as follows:

(i.) Hyperperistalsis of the duodenum with regurgitation from second to first stage.
(ii.) Hyperperistalsis of the stomach.
(iii.) Hypermotility of the stomach often followed by a slow rate of emptying.
(iv.) Hypermotility of the intestine.
(v.) Tenderness on palpation over the cap.
(vi.) Initial pyloric spasm.

Direct Signs.

The Presence of a Crater.—The method of detection of a crater has been described. The appearances enumerated provide the most certain X ray evidence of the presence of an ulcer. The size of craters, seen radiologically, varies from dots not more than two millimetres in diameter to objects as large as a threepennypiece. The accessory pockets of chronic perforating ulcers appear as excrescences from the walls of the cap and once filled their presence may be detected for twenty-four hours or more.

Deformity of the Cap.—Almost invariably when a duodenal ulcer is present some irregularity is present in the shape of the cap. In recent ulcers the deformity noticeable is usually an incisura in one or more borders of the cap and it is generally spasmodic in type. An incisura may be very small and is frequently difficult to detect, especially when it is situated on a limited area of the anterior or posterior wall. In such positions it can be seen only when the patient is rotated.

In chronic ulcers the deformity is usually more gross; the borders of the cap appear as serrated edges and its lumen is encroached on by the indentations. Such deformities are usually due to scar contraction of an old ulcer and frequently no crater can be detected with certainty. In this connection it is interesting to note W. J. Mayo's pathological description of such ulcers:

> The mucosa of the duodenum is thin, smooth and granular and chronic duodenal ulcers may not therefore have the characteristics we have learned to expect from experience with gastric ulcers. I have excised a number of duodenal ulcers in which there was considerable scar-tissue in the submucosa and muscularis, and marked evidence of localized peritonitis; yet the actual ulcer was a mere slit or dimple surrounded by an eroded discoloured patch of mucosa.

Indirect Signs.

Hyperperistalsis of the Duodenum.—Hyperperistalsis of the duodenum with regurgitation from the second to the first stage is the most useful of the indirect signs. Its detection is certain only on the fluorescent screen, but it may sometimes be demonstrated on a film as a blurring of the shadow caused by rapid movement of the barium during the exposure.

Hyperperistalsis and Hypermotility.—Hyperperistalsis and hypermotility followed by sluggish peristalsis and a slow rate of emptying of the stomach are useful secondary factors, the significance of which should not be overrated. The motility of the stomach shows such wide variations within normal limits that its utility as a point of evidence is limited. Hyperperistalsis is usually more reliable and when present in combination with other signs it forms a useful guide.

Hypermotility of the Intestine.—Hypermotility of the intestine is an interesting observation in many cases of duodenal ulcer. Normally the head of an opaque meal appears in the region of the hepatic flexure six hours after ingestion, whereas it may reach the descending colon in the same time if a duodenal lesion is present.

The limitations of this factor as dependable evidence obviously depend on the exclusion of any irritative condition in the intestine.

Tenderness.—Tenderness over the duodenum is of variable occurrence and of minor importance. In some cases tenderness on pressure over an ulcer may be accurately elicited, but in such instances one of the direct signs is almost invariably present, so that the actual tenderness is rather of incidental interest than of diagnostic importance.

Initial Pyloric Spasm.—Pyloric spasm in the early stages of the examination is of frequent occurrence and may cause a delay of several minutes before any barium enters the duodenum. Gentle abdominal massage with the patient lying on the right side usually results in relaxation of the spasm.

Fallacies.

The position of the duodenal cap in the right upper quadrant of the abdomen and its proximity to such important surgical structures as the gall bladder, common bile duct and pancreas render it necessary that a differential diagnosis of lesions of these organs should be made as far as is possible.

Inflammatory and infective lesions in and around the gall bladder frequently give rise to radiological signs similar to those found in duodenal ulcer. Bands of adhesions give rise to deformity of the cap closely resembling scar contraction of an ulcer and spasmodic incisuræ are frequently seen in both conditions.

The imprint of an enlarged gall bladder on the cap produces a pressure defect which is characteristic, usually crescentic in shape and smooth in outline. It is therefore unlike any deformity produced by an intrinsic duodenal lesion.

Duodenal diverticulum is a rare condition which may be confounded with an ulcer. When it occurs, however, it is usually situated beyond the cap.

Of the possible pitfalls mentioned undoubtedly the most common in occurrence are those associated with gall bladder lesions and a definite opinion as to the actual site of the lesion, based on the radiological findings, is often impossible. It is in such cases that a combination of several of the indirect signs may prove of valuable assistance.

Results.

The proof of the accuracy of any diagnosis has in the operating table and *post mortem* room two reliable courts of appeal. In the present instance the most satisfactory method of acquiring results is provided by operative findings.

In large institutions, such as the London Hospital, Manchester Royal Infirmary and the Mayo Clinic (United States of America), where the technique described is employed, it is found that in the patients who come for operation the radiological diagnosis is correct in over 90%.

The actual statistics of the hospitals mentioned provide interesting comparisons and indicate a high degree of consistency of the method in the hands of the men attached to each.

In a small series extending over the last nine months a radiological diagnosis of duodenal ulcer has been made in thirty-two cases. Twenty-three patients have been operated on and in three the opinion has been incorrect. In one instance the X ray signs were indefinite and an undecided opinion was given. In the second case a definite diagnosis of a small duodenal ulcer was made and none was found at operation nor could an explanation be formulated of the appearances noted. Thirdly an opinion was given that no ulcer was present and at operation a lesion was found on the anterior wall of the duodenal cap.

Conclusion.

Since the discovery of X rays in 1896 ever increasing experience has proved that radiology must become a very important link in the diagnostic chain and probably in no section of the subject has more progress been made than in the recognition of gastro-intestinal lesions.

The debt of gratitude which we owe to the pioneers of the subject cannot be overestimated. The wealth of experience which has been placed on record in such a comparatively short time, is astounding.

Many of those early workers were physicians and surgeons who correlated their own clinical and radiological findings when compounding their diagnoses and in this they have set an example worthy of emulation. For now that the subject has become a special branch of medicine, how much more essential is it that the closest cooperation should exist between the physicians and surgeons on the one hand and the radiologists on the other?

It is only by such cooperation that we can hope to obtain the best results from our comparatively

new science and it is only by correlation of all the evidence at our disposal that we shall be able to compare successfully organic lesions within the human body with phantoms seen in a dark room or shadows on a photographic film.

LIPIODOL INJECTIONS AS A DIAGNOSTIC AGENT.

By J. F. MACKEDDIE, M.D. (Melbourne),
Physician Attending In-Patients, The Alfred Hospital, Melbourne.

THE following are spinal cases that were investigated by the lipiodol method introduced by Sicard.

Lipiodol, a solution of iodine in poppy oil, it may be explained, was the result of Sicard's search for a substance that combined three properties. It had to be opaque to X rays of greater specific gravity than cerebro-spinal fluid and innocuous to nerve tissue.

Lipiodol was found to possess those properties and its injection into the cerebro-spinal fluid *viâ* the *cisterna magna* was successfully used by Sicard, Sir James Purves Stewart, Sargent and others in localizing obstruction in the theca.

I have refrained from giving full neurological findings, and have emphasized only the points that would lead to a better appreciation of the part played by lipiodol in the localization of the lesion.

CASE I.—Mrs. L., *ætatis* forty-six, for many years a seamstress at the Alfred Hospital, suffered four years ago from numbness in the right thigh and then in the left, succeeded by weakness of both legs. There had never been any pain at any time nor bladder symptoms. Examination at that time revealed only the loss of the right abdominal reflex. Subsequent examinations revealed the classical signs of an advancing spastic paraplegia ending in almost complete paralysis with definite sensory phenomena. No compression was suspected.

The upper limit of sensory disturbance was ill-defined. A pin prick was not recognized as such below the lower border of the second lumbar segment. Between that line and the twelfth dorsal dermatome it was felt as possibly something sharp. This was as nearly as possible alike on both sides. There was no suggestion of a hyperæsthetic area. Limits of effective tactile sensation were much less easily defined. The posterior column conduction was greatly diminished.

Only the lower abdominal reflexes were abolished. Beavor's sign was not noted. Lipiodol injected through the *cisterna magna* was held up at the level of the eighth vertebra (see Figure I.).

Examination after its introduction gave the following results. There was now a band of very great hypersthesia running along the tenth to the eleventh dermatomes, while the more or less indefinite delimitation of sensory disturbance became more definite. Mr. Maclure, who saw the patient at this stage, put two questions to her: "Have you or had you at any time any pain?" "No!" "Have you had any trouble with your water?" "No!"

The absence of these classic symptoms of spinal compression greatly discounted the lipiodol findings in his mind and he consented to explore with some misgivings. The eighth and ninth thoracic laminæ were removed and the lipiodol demonstrated. A small smooth tumour lay on the left latero-posterior aspect of the cord without involving any of the nerve roots. Its removal was a matter of great difficulty and anxiety for it was apparently incorporated in the substance of the cord. Fears were entertained at the operation that considerable damage was done to the cord. The condition of the patient for some weeks afterwards seemed to confirm our fears, the condition being as nearly as possible that of a total transverse lesion with complete retention of urine and profound anæsthesia and lost deep reflexes. However, there soon appeared signs of improvement and six months after the operation she was walking about with the assistance of somebody's arm and reported that she was getting steadily better.

Mr. Trinca's note on the tumour is to the effect that it had the structure of a typical psammoma.

CASE II.—Nurse R., a graduate of the Alfred Hospital, was seen some four years ago for "nerves." She had severe pain in her back and complained of being "run down."

No neurological findings were discovered at that time. Two years later she complained of pain in her back and down the right thigh; there was undoubted rigidity of the lower extremities. Abolition of the left abdominal reflex was the only pathological finding. In the middle of 1925 she manifested great spasticity with ankle and patellar clonus and great difficulty in walking; both abdominal reflexes were absent and there was definite bladder and rectal trouble.

There was no alteration of sensation of any kind anywhere.

Dr. Sewell saw her with me at this stage and in view of the fact that there was some upper limit indicated by the persistent pain that had been a very early symptom, and of the onset of retention symptoms, we felt that lipiodol was indicated even if there were no Froin's syndrome of loculation and an absence of all sensory phenomenon. The result is shown in Figure II., in which the lipiodol is seen resting after an unobstructed course down the theca in the *cul-de-sac*.

She was being investigated at the same time as Mrs. L. and the pair made a very interesting study.

The surgeon's inquiry as to bladder symptoms and pain would have been answered in her case in the affirmative and yet there was no compression.

CASE III.—H.W., *ætatis* forty-six, a salesman, gave a history of twenty months' pain in the back and legs more severe posteriorly. All the varieties of pain were described by him as "stabbing," "dragging," "numbing" and "paralysing." He passed his nights standing at the head of his bed gripping the head rail for support till his fingers had actually developed corns. There were no bladder or rectal symptoms.

Repeated examination revealed a complete absence of any neurological abnormality, either motor or objective sensory or reflex. Lumbar puncture yielded fluid with all the evidence of an undoubted Froin's syndrome, indicating a thecal obstruction. An attempt was therefore made at localization from symptoms alone. There was an area not encroached upon by pain; that was the region corresponding to the distal (that is on the back of the thigh, the upper) half of the second sacral distribution and the perianal area of the third, fourth and fifth sacral and coccygeal. This exempted the contribution of the *conus* to the *cauda* and thus marked off very definitely the lower limit of the obstruction. Pain in the back was as high as the first lumbar distribution. This, of course, could be caused by compression of the lumbar distribution at its exit from the theca, in its course in the theca, or at its actual separation from the cord itself. The upper limit was much less definite; nevertheless as the lipiodol shows, a very fair guess at the position was possible. Lipiodol injected through the *cisterna magna* came to rest at twelfth thoracic to the first lumbar vertebra (see Figure III.).

Examination after the lipiodol was in the cerebro-spinal fluid made more certain of the painful areas above described, as the pain became intense, paroxysmal and very sharply defined. But no objective neurological phenomena could be demonstrated.

An operation was indicated. The cord was exposed. the lipiodol retrieved and a small soft hæmorrhagic growth removed. It lay with its lower end just reaching the *conus*, wrapper round by the *cauda* and attached by two large polar vessels to the *pia arachnoid*. It was easily removed leaving the structures intact. Apparently it was too soft to cause compression signs.

For the first time for nearly two years the patient passed a peaceful night in bed and his subsequent recovery was complete.

This patient was referred to me for lipiodol investigation by my colleague, Dr. A. V. M. Anderson, who has kindly given me permission to publish his notes of the following case. The tumour was removed by Mr. Brown.

Mr. Trinca's report is to the effect that the tumour consisted of a number of large blood spaces lined by tumour cells arranged in a diffuse manner without any definite intercellular substance. The vascular element was extensive enough to justify the name of angioma. Individual cells in shape and arrangement resembled carcinoma rather than sarcoma. The matrix was represented by a lowly nucleated form of fibrous tissue.

The diagnosis was in consequence angio-endothelioma.

CASE IV.—Mrs. K. suffered from a condition very similar to the preceding patient H.W. Repeated examinations failed to disclose any objective neurological findings and yet the pains in her back and legs had been more or less continuous and severe for six years. During the last six months the pain had been much intensified so that sleepless nights, even with sedatives, were the rule.

The cerebro-spinal fluid yielded evidence of a typical Froin's syndrome which is almost pathognomonic of obstruction. An attempt to localize the obstruction from symptoms only, as in the last case was interesting. Unlike the case of H.W. there was no area of sacral distribution free from pain, that is the lowest components of the *cauda* were involved and one would expect the block to be located lower down. Pain reached as high as in H.W., that is at the first lumbar distribution. Therefore, although the same indefiniteness obtains concerning the upper limit, we are enabled to say that the obstructing body is longer and its probable location is at or just below the *conus*.

It is to be observed here that in spite of the fact that the conal component of the *cauda* was involved, there were no bladder or rectal symptoms.

Immediately on the introduction of the lipiodol the upper limit of the painful areas was strikingly picked out by an acute exacerbation of the pain. While this upper painful area was somewhat more sensitive to handling, there was nevertheless no abnormality in the way of anæsthesia below this line or hyperæsthesia above it.

The lipiodol injected through the *cisterna magna* came to rest at the first lumbar vertebra (see Figure IV.).

With the guidance of the lipiodol picture the lamina of the first lumbar vertebra was removed and the lipiodol released. The actual obstruction lay under the lamina below; on its removal a tumour very similar in appearance to that of H.W. was disclosed. It was packed tightly among the components of the *cauda*, its upper limit just reaching the *conus*; it was a little difficult to deliver, but was unattached to any cord structures except for two large vessels. It was much longer and stouter than the preceding tumour, yet in spite of its tight fit in the theca no compression signs had manifested themselves at any time.

For the first day or two there were severe pains in both feet, but on the fifth day after the operation the condition was more or less normal and there had been no sign of the old pain, so that a complete recovery is confidently expected.

This patient was also referred to me by Dr. A. V. M. Anderson and again Mr. Brown was the surgeon.

Mr. Trinca, our pathologist, made notes to the effect that the tumour consisted of cells arranged as in the common so-called dural-endothelioma; although the tissue was highly vascular, the blood was enclosed in definitely walled vessels, contrasting strongly with that of H.W. In places there was a tendency to concentric disposition of the cells round the vessels suggesting a peritheliomatous nature.

CASE V.—Mrs. T., *ætatis* forty-five years, began seven years ago to notice pain in the right groin and down the front of the thigh, followed soon by a similar condition of the left side; stiffness of her legs supervened, then dragging of her legs and walking only with help and finally complete paralysis with retention of urine. The bladder later emptied itself automatically and incompletely. Examination revealed a condition of spastic paraplegia with the legs in slightly flexed position. On the right side there was complete sensory loss up to level of second lumbar dermatome and comparative loss as high as the twelfth dorsal. On the left side all forms of sensation were much diminished, pain and the temperature sense alone were lost at the third lumbar segment. The posterior columns were comparatively intact; joint and vibration sense was almost normal.

A Wassermann test applied to the blood serum and the cerebro-spinal fluid did not yield a reaction. Lipiodol injected through the *cisterna magna* was held up at the tenth to the eleventh thoracic vertebra (see Figure V.). When the theca was exposed by Mr. Hurley it was found that the *dura mater* and leptomeninges were bound down over more than three centimetres of the cord in the form of a hard ring. An attempt was made to dissect this off, but was only partially successful.

As usually happens there was a deepening of symptoms; for example complete retention returned and the sensory loss on the left side deepened to the degree of the loss on the right, but at the end of a fortnight the condition was practically what it was before the operation. The woman returned home to the country and the last accounts were of slow but definite improvement. Needless to say no over sanguine hopes are entertained for this patient.

This patient was referred to me for lipiodol investigation by Dr. Sewell to whom I am indebted for the notes and permission to publish the details.

Dr. Mollison reported on the compression tissue that it was non-specific inflammatory products.

CASE VI.—B.E., a labourer, *ætatis* thirty-five, was admitted for a condition diagnosed as a meningo-myelitis of doubtful specific origin. There was a history of a somewhat sudden onset six months previously of pains across the abdomen and in the lumbar and sacral regions. Then followed wasting of the lower extremities and accompanying weakness. There was also rectal and bladder incontinence. Examination revealed a distended abdomen with absence of the upper and lower reflexes; all the deep reflexes of lower extremities were abolished. There was considerable disturbance of effective tactile sensation and of pain and temperature sense, the upper limit of which at this stage was quite indefinite. The posterior column conduction was affected to a much less extent. Beavor's sign was elicited (the umbilicus moved upwards). Just then a rapid change took place in the area involved; the upper limit receded downwards and the lower limit upwards so that all abdominal signs and symptoms vanished and the rectal and bladder trouble abated. A careful examination at a later date revealed the following sensory loss. On the right side the recognition of a pin prick was dulled from the level of the middle of the third lumbar dermatome, as far as the middle of the first sacral where it became normal again. On the left normal recognition of pin pricks was dulled at the lower limit of the third lumbar, became absolutely lost at the lower limit of the fifth lumbar as far as the beginning of second sacral, where it became normal. Effective tactile sensation followed similar but much less definite limitations. The cerebro-spinal fluid yielded a definite loculation syndrome. Lipiodol was given by the *cisterna magna* route. The level it took up was not compatible with the latest neurological findings; it stayed at the level of the seventh thoracic vertebra (see Figure VI.). But a curious thing happened. The upper limit of the disturbance took up its former position; the abdominal signs and symptoms returned with all the old pain and the patient said he was as bad as he had been at first. But there was a quick subsidence of all abdominal phenomena, for the lipiodol in a day or two found its final resting place at the twelfth thoracic vertebra and so fitted in with the final clinical findings (see Figure VII.).

Mr. Kennedy exposed the cord over the lipiodol and retrieved the latter. There were dense adhesions everywhere which were freed as far as could be from the caudal tissues. From the nature of the onset and history we were not surprised to find no obstructing growth. There followed distinct improvement, but dramatic results in a case of this kind are not to be expected. Needless to say on the finding of a reaction to the Wassermann test

vigorous antisyphilitic measures had been taken and it was only when there was a fixed residue and a definite Froin's syndrome that operative measures were considered. The suspended lipiodol decided the matter.

CASE VII.—H., *ætatis* sixty-eight, was an agent. On admission his right knee jerk was exaggerated; his left knee jerk was absent. His right ankle jerk was absent; his left ankle jerk was exaggerated. There was a distinct sensory loss over the front of the left thigh which was the seat of intense pain and of considerable wasting.

On the right side the wasting was in the posterior aspect of thigh, affecting chiefly the gluteal region. There had been very great pain down back of thigh and leg with occasional recurrence. There had been a history of a "sciatica" of the right leg some twelve months before and as it disappeared to a large extent, a similar condition supervened in the right thigh, but being anterior it was not called "sciatica." The sensory distribution on the front of the left thigh showed it to be confined entirely to femoral distribution. A diagnosis of a peripheral lesion was made which did not call for even a lumbar puncture. But the patient had been sent in by a surgeon for a lipiodol injection and examination.

The result is shown in Figure VIII. The lipiodol meeting with no obstruction has sunk into the little *cul-de-sac* in the theca somewhere in the neighbourhood of the second sacral vertebra.

Our records at the Alfred Hospital include the histories of five more patients; of these brief mention will suffice. One man and one woman had more or less classical signs of compression. In the former the upper limit of signs could be defined with some accuracy; in the woman this was not so. In both Froin's loculation syndrome was determined and in both the lipiodol rested very definitely at a certain level of the theca. Both refused operation.

In three other men with signs and symptoms of compression the diagnosis of possible meningomyelitis was made. Froin's syndrome was absent in all three. Lipiodol made its way at once to the terminal *cul-de-sac* and thus eliminated compression.

In these patients only one lipiodol injection was made in each with the exception of B.E. Some time subsequent to the *cisterna magna* injection, more lipiodol was given by the thecal route below the seat of obstruction. This was allowed to run up to meet the obstruction as indicated by the *cisterna magna* injection and a skiagram taken in the inverted position. It certainly defined the lower limit of the obstruction, but it was considered unnecessary in subsequent cases.

Conclusions.

1. In all cases of spinal compression localization is difficult and the lipiodol method makes a certainty of a doubtful diagnosis.

2. Some cases of compression may be impossible of localization without resort to lipiodol. It is questionable if a spinal compression has ever been diagnosed on subjective phenomena alone.

3. While the main value of lipiodol lies in exact localization of compressions, its assistance in excluding possible compressions cannot be ignored.

4. In no case did a lipiodol injection indicate obstruction in the absence of a Froin's syndrome. Broadly speaking, a lumbar puncture can eliminate a compression.

5. While lipiodol may temporarily irritate nerve tissue that is abnormal, it seems to have no ill effect on healthy tissue and certainly it seems to rest innocuous enough in the terminal *cul-de-sac*.

6. Besides the important fact that the field of operation can be greatly curtailed owing to accurate localization, the assistance and encouragement to the surgeon who has before his eye the outlined obstruction as his objective, must be very great.

7. When the obstruction is accurately placed by means of lipiodol, careful examination of a number of patients in whom definite sensory findings are in evidence, will lead to a better understanding of segmental levels.

Thus total analgesia at the second lumbar dermatome is caused by a block at the level of the eighth thoracic vertebra. This can only be explained on the assumption that such an area as the second lumbar dermatome is represented at higher levels of the cord as well as in the second lumbar segment, by means of stray afferent fibres that get into the cord *viâ* higher roots. In computing segmental distribution, we have to add two to three vertebral body lengths over and above the two to three vertebral body lengths added on account of the fact that the issuing nerves have traversed that length in the theca before emerging from the canal.

I am speaking now of the line of total abolition of a particular sensation.

If we measure from the upper limit of the partial loss or diminution, that is from the first return of normal sensation, we have, of course, only to add the length the nerve has travelled in the theca before emergence at the intervertebral foramen.

Reports of Cases.

A CASE OF CENTRAL RUPTURE OF THE PERINEUM.

By A. G. E. NAYLOR, L.R.C.P., L.R.C.S. (Edin.),
Loch, Victoria.

AFTER being summoned to attend a *primipara*, aged thirty-six years, I arrived when the child's head was pressing on the perineum. Pains were strong and I used chloroform at once and discouraged muscular efforts. The perineum began to be stretched unduly in an anteroposterior direction. Soon I noticed a bleeding spot near the anus. With the next pain this spot became an undoubted rupture through which *liquor amnii* spouted followed by protruding membrane. There was every likelihood of a complete tear. With the scissors I slit the vulval opening backwards as far as the rupture (episiotomy) and with the following pain the baby was born. It weighed 4·2 kilograms (nine and a half pounds). The anal sphincters and the bowel were untorn. The patient went home at the end of a fortnight with a repaired and useful perineum. After many years of practice these were new experiences for me. The assisting nurse who had had a long experience of midwifery in England, had never seen such a case. In Herman's "Difficult Labour" (1906 edition) there is a quaint illustration of the condition. Perhaps if I had seen the patient earlier, I might have been able to prevent the accident, perhaps not.

AN ODONTOME FROM A BULLOCK.[1]

By J. BURTON CLELAND, M.D., Ch.M. (Sydney),
Professor of Pathology, The University of Adelaide, Adelaide.

I WISH to show, on behalf of Dr. Watson, of Quorn, and myself with permission of the owner, Mrs. Ellis, a very remarkable ivory-like mass weighing 5·1 kilograms (eleven and a half pounds) whose nature had given rise to considerable speculation, but which was unquestionably an odontome. The mass was found "in the carcase of a bullock" (presumably by this is intended "amongst the bones of a bullock") at Mr. McFarlane's station on the River Murray by a drover and was given to Mr. Taylor before the year 1863. From its remarkable shape, resembling to some extent a heart with auricles and ventricles, the specimen was for long considered a fossilized bullock's heart. Eminent geologists have inspected it and considerable speculation has arisen from time to time as to its nature. The specimen is naturally very much valued by its owner and it is to be hoped that it will eventually find its way to the University Pathological Museum where it may be available for inspection by anyone interested. The mass is really a huge odontome. Ground sections of very small fragments show that it is composed of cement, cement lacunæ with fine canaliculi being numerous and lamination being present. It is possible, of course, that in other parts dentine and enamel may also be present. The growth is probably chiefly, if not entirely, a cementome, however.

I have seen somewhere, but have not yet been able to trace the source, an illustration of a somewhat similar specimen. Bland Sutton, in "Tumours Innocent and Malignant" figures various odontomes and the only one somewhat resembling this specimen is a cementome. The size and weight of the tumour probably interfered with the animal obtaining nourishment in sufficient quantities and so was probably responsible for its death.

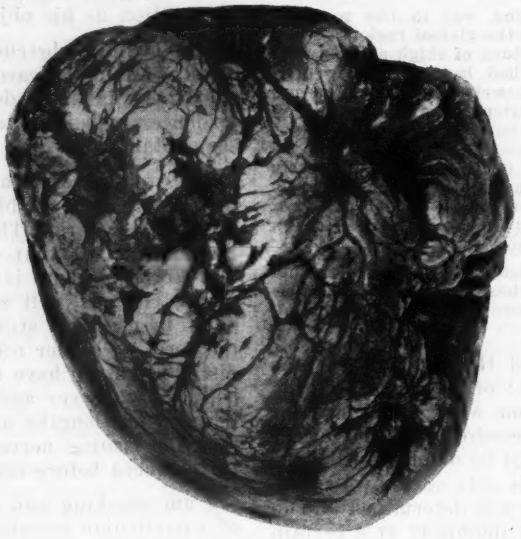

FIGURE I.

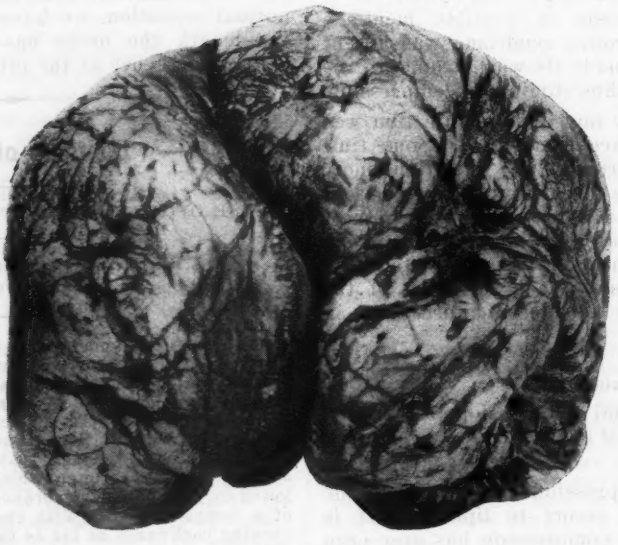

FIGURE II.

In conjunction with Dr. Watson I have described the appearance of the tumour as follows: A somewhat rounded tumour presenting a brain-like appearance with "convolutions" flattened in places, in others with the sulci exaggerated and with an irregular longitudinal fissure passing two-thirds round the circumference. The longitudinal fissure passes through the middle of the base of the "brain" where there is an irregular, fairly wide and deep depression. The appearance might easily be interpreted also by the laity as a highly abnormal fossilized heart. Its longest diameter is 21·25 centimetres (eight and a half inches), the shortest diameter 16·25 centimetres (six and a half inches) and the height 17·5 centimetres (seven inches). The circumference measured in various directions, varied from sixty-two centimetres (twenty-four and a half inches) to 57·5 centimetres (twenty-three inches) and to fifty-one centimetres (twenty and a half inches). Its weight was 5·1 kilograms (eleven and a half pounds), the density 1·889 and it displaced 2·7 litres (ninety-seven and a half ounces) of water. The whole surface was polished, presenting a dull ivory colour, in most places obscured by a smoky brown. A few artificial cracks reticulated a small portion of the surface. On two or three prominent positions are polished flattened facets of small size. Where a piece has been broken off a dense white structure resembling dentine is revealed.

Odontomes are not uncommon in horses, especially in the temporal region and it is possible that the finder may have mistaken the bones of a horse for those of a bullock. If the animal was a horse, however, one would hardly expect the tumour to have been allowed to grow to such an incapacitating size in the days when horses were scarce and received careful attention.

[1] Read at a meeting of the South Australian Branch of the British Medical Association on March 25, 1926.

AN UNUSUAL CASE OF TYPHOID FEVER.

By K. C. ROSS, M.B., B.S.,
Senior Resident Pathologist, Melbourne Hospital.
(From the Walter and Eliza Hall Institute for Research in Pathology and Medicine, Melbourne.)

THIS case is of interest since at no time during the disease was any immunological response evident as judged

by repeated agglutination tests, but was regarded clinically as a case of typhoid and proved to be so at autopsy.

A female, *ætatis* thirty-seven years, was admitted on February 24, 1926, ten days after the onset which had occurred insidiously with headache, increasing deafness and head noises. A single, slight epistaxis had occurred and there had been some diarrhœa. On admission she was in a confused mental state, with a temperature of 38·5° C. (101·4° F.) and a pulse rate of 105. The abdomen was distended and tender; there were "rose spots," but the spleen was not palpable. The leucocyte count was 4,000 per cubic millimetre. On February 26, 1926, there was some melæna and a few days later signs of infection developed at the base of the right lung. Her temperature remained high, ranging between 38·3° and 40·5° C. (101° and 105° F.) till her death on March 5, 1926, after an illness of nineteen days.

Culture tubes were inoculated with blood, stools and urine taken during life, but no typhoid bacilli were grown; no reaction was obtained to the Widal test throughout the illness.

Three hours after death material was obtained from the gall bladder and spleen for bacteriological examination, but an autopsy was not performed until five hours later. In the ileum were several but not very numerous ulcers which for the most part lay in the transverse axis of the gut but were otherwise typical. There were gross, ragged ulcerations at the ileo-caecal junction and a few small ulcers in the caecum and ascending colon. The mesenteric lymphatic glands were very enlarged, soft and suppurating. The spleen was soft, but not enlarged; it weighed 142 grammes. The middle and lower lobes of the right lung had consolidated and were breaking down.

From the gall bladder, spleen and mesenteric lymphatic glands there were cultured motile, Gram-negative bacilli which did not ferment lactose, but yielded acid without gas in glucose and mannite and which were agglutinated with standard *Bacillus typhosus* (Penfold) to 1 in 8,000, but were not agglutinated in any dilution with serum for *Bacillus paratyphosus A* or *B*.

I am indebted to Dr. F. B. Lawton for permission to publish the particulars of this case.

Reviews.

TREATMENT.

THE fact that the "Index of Treatment" edited by Dr. Robert Hutchison and Mr. James Sherren with the help of many contributors, has entered its ninth edition is in itself substantial proof of the lasting popularity of this great work.[1]

All the articles have been revised and several rewritten. Three subjects hitherto omitted appear for the first time, namely infective endocarditis, spinal analgesia and surgical tuberculosis. Horder, the writer on this fatal form of endocarditis, divides the treatment into preventive and curative measures. In the former he holds that special attention should be drawn to dead or suspicious teeth, septic tonsils and those parts of the alimentary canal which are known to harbour causative streptococci; for the causative measures, as he considers this disease to be an arterial pyæmia, he advocates fresh air, sunshine and good feeding; he also employs whole blood transfusion. For the time at least he considers the latter helpful and therefore it should be tried. Antisera he finds disappointing and chemotherapy useless. Of sensitized vaccines he speaks more hopefully. The article on diabetes which is very explicit, will be read with interest by the enterprising country practitioner. Dr. Sprigg goes fully into the question of diet. He divides his cases into the mild and more severe, giving suitable diets for each. He gives minute instructions as to the administration of "Insulin" and in concluding asks the question: "Can 'Insulin' be safely given in general practice?" His answer is emphatically: "Yes!"

The article on fractures will be useful to the general practitioner as all the salient points in the modern methods are given. Gonorrhœa and syphilis, both by the same author, are well written. In the former one thing is lacking. Abrahams, the writer, does not express in clear and definite language what he means by the term "cure."

Tropical diseases are considered by Manson-Bahr. That important disease yellow fever is dismissed curtly. The epoch-making investigations of Noguchi in central South America are mentioned, but not commented on.

The two articles on asthma are on the whole disappointing. The cutaneous test, advocated in England and boomed in America, have not fulfilled their promise; in fact there is just a possibility that they may in very many cases have clouded the essential factors which are on the one hand a vitiated metabolism and an irritable bronchial centre on the other.

In this new edition the different forms of physical treatment have been replanned and freshly written; a new article on psychotherapy has been added. Psychotherapy when reduced to its ultimate issues really means personality; notwithstanding this in these modern days psychic methods have been carefully investigated and scientifically employed in treatment in the form of suggestion, hypnotism and psychoanalysis; their usefulness has been demonstrated largely in affections resulting from the stress and strain of war. On these neuroses Dr. Crichton Miller writes very clearly and gives the practitioner a well considered, sensible *résumé* of Freud's doctrine. In the treatment of some of these neuroses hypnotism is ably discussed by Dr. Milne Bramwell; he refers to the so-called dangers of this method. He states that the employment of hypnotism by medical men acquainted with the subject is absolutely devoid of danger.

The articles on the surgical diseases of the prostate, kidney and gall bladder have been entirely revised and brought up to date.

Many of the older articles have been read with interest and even in those valuable additions have been made, notably on pulmonary tuberculosis by Sir Robert Philp, in which he gives a concise rendition of modern opinion and the results of his own experience which has been extensive. Contrary to the views of many other specialists he still retains his faith in tuberculin and favours that of Béraneck on account of its supposed greater potency and lesser toxicity than some of the others. In this article a concise description of artificial pneumothorax is given, while the scanty reference to the surgery of the thorax in pulmonary tuberculosis causes great disappointment.

It is a difficult task in reviewing a well devised work of this kind to discriminate what should be commented on and what should be left out, as it is so full of many good things. We, therefore, without hesitation recommend it to all practitioners of experience as a book of reference and also to the younger men who are just embarking on their career, more especially if they elect to go in for a bush practice. In the "Index" they will find everything ready to hand and can face more ably any emergency in medicine, surgery and all the specialities with confidence in the fact that all is described concisely in this admirable book; even the almost forgotten art of prescribing is not omitted. This subject is excellently treated by Dr. James Calvert who gives good results as to prescription building and the pitfalls of incompatibilities in the compounding of medicines, often neglected in modern medical schools. However much it may be ridiculed by some, the gentle art of prescribing well is a very important element in successful medical treatment. To quote Dr. R. Hutchison, the editor: "There was a time undoubtedly, when they (medicines) were used too much, but there is now a tendency in some quarters to use them too little. Perhaps the truth as to their value is expressed in the statement that they sometimes cure, often relieve and always console."

[1] "An Index of Treatment," by various writers. Edited by Robert Hutchison, M.D., F.R.C.P. and James Sherren, C.B.E., F.R.C.S.; Ninth Edition, Revised and Enlarged. 1925. London: John Wright and Sons Limited. Royal 8vo., pp. 1035. Price: 42s. net.

Before closing it would be ungracious to pass by the introduction entitled "Some General Principles of Therapeutics" penned by Dr. Hutchison, without commenting on the sound principles given and the charming English in which they are expressed.

INFANT FEEDING AND NUTRITIONAL DISORDERS.

THE fourth edition of "Feeding and the Nutritional Disorders in Infancy and Childhood," by Dr. Julius H. Hess, is to hand.[1] Much of the previous edition has been rewritten and important new chapters have been added, making the book considerably larger than the modest first edition which appeared in 1918.

Part I. is taken up with the discussion of general considerations, such as the anatomy and physiology of the digestive tract and metabolism in infants. In Part II. breast-feeding is considered together with wet-nursing and the nutritional disorders in the breast-fed infant. The author's opinion is that the young mother of today is better able to suckle her offspring than was her sister of fifteen or twenty years ago. He emphasizes the fact that breast-feeding during the first two or three weeks of the infant's life is carried on under unfavourable conditions which do not indicate the possibilities of the breast as a secreting organ. When the customary life and daily habits are resumed, lactation may be carried on with success in spite of difficulties in the early days. Contrary to most modern teaching he recommends the practice of substituting one bottle-feeding daily for one breast-feeding, at the end of the third or fourth month to relieve the mother and to train the baby to take the bottle. Experience shows that, especially if five feedings in twenty-four hours are given, the substitution of a bottle-feeding for one of these invariably reduces the supply of breast-milk. The lack of sufficient stimulation of the breasts leads to a decrease in the secretion of milk and to early weaning. The feeding of the premature baby is adequately described.

Part III. is devoted to the consideration of artificial feeding. In this section the author after referring to other methods of adapting cow's milk to the requirements of the infant, describes that one which he has found to be the most satisfactory, namely milk mixtures with carbohydrate added. He establishes a basis for the preparation of diets which maintains an absolute relationship between the quantities of the individual food elements of the diet and the requirements for the growth and nutrition of the infant per pound of body weight irrespective of the size and frequency of the feedings. The estimation of the caloric value of the food is used as a check on under-feeding or over-feeding. He lays stress on the fact that in the application of rules for the feeding of normal healthy infants it must be remembered that each infant must be fed to meet its individual requirements and the rules modified to meet the demands of the individual. The amounts of the food elements needed as a minimum by the normal healthy infant are estimated as follows: For each kilogram of body weight 3·3 grammes of protein, 3·3 to 4·4 grammes of fat and 6·6 grammes of carbohydrate. This amount of fat and protein is contained in approximately one hundred cubic centimetres of milk. To this is added sugar and starch to make up the required amount of carbohydrate. Water is added to make up the volume of the food. Orange juice and cod liver oil are added to the diet from the second to the third month. Diets suitable to infants from six months to six years are given as well as a very complete list of food values of individual servings.

The rest of the book is taken up with descriptions of the nutritional disorders which occur in artificially fed infants, including rickets, scurvy, tetany *et cetera*.

This book is an exceedingly good and clear exposition of the present ideas on infant feeding and nutritional disorders. The author has made use of his own clinical experience and is not dependent on the opinions of others. It should prove a valuable addition to the library of the general practitioner and specialist alike.

[1] "Feeding and the Nutritional Disorders in Infancy and Childhood," by Julius H. Hess, M.D.; Fourth Revised and Enlarged Edition; 1925. Philadelphia: F. A. Davis Company. Demy 8vo., pp. 556. Price: $4·50 net.

A BOOK FOR MOTHERS ON INFANT FEEDING.

IN "The Mothercraft Manual" Miss Mabel Liddiard has discussed the questions dealing with the first two years of infant life in a manner that any mother can understand.[1] An exception to this statement must be made in regard to the much debated question of artificial infant feeding.

The whole book is an outline of the principles taught by Sir Truby King who is the founder of the New Zealand Mothercraft Training Society. The chapter on "Natural Feeding" deserves the strict attention of every mother and does Miss Liddiard great credit.

She lays stress on many practical points which a number of medical men could read with advantage.

In discussing the question of weaning she states that the ideal is to give the baby nothing but breast milk for the first nine months and then to take five weeks over the weaning. Some American authorities prefer to begin by adding food to the infant's diet in addition to the breast at about the sixth month. Well cooked cereals before one or two nursings at the sixth month followed a few weeks later by carefully prepared vegetables and broths, is said to have proved very beneficial.

Miss Liddiard does not discuss the question of the baby that will not take a bottle when mixed feeding is begun. This is extremely common, is peculiarly annoying, not without embarrassment to normal progress and at times even dangerous. In the chapter on "Artificial Feeding" the principles recommended are those of Sir Truby King who modifies cow's milk and adds New Zealand cream. The inclusion of cocoanut oil has been subjected to some adverse criticism. The feeding recommended can be easily followed by a person trained in these methods, but not by a mother who is a stranger to them. It is, of course, possible to feed infants even more satisfactorily by other less complicated methods.

Such statements as appear on page 70 in reference to starch are at variance with present knowledge: "Until the baby is six months old all carbohydrates should be given in the form of sugar, because the digestive ferment which deals with starch is absent until then and such additions to the food give indigestion, colic and wind."

In view of the many mistatements and in view of the unreserved acceptation of the Truby King method, we cannot recommend this work as a book of reference for mothers.

DISEASES OF THE EAR, NOSE AND THROAT.

THE time and energy devoted by Dr. Gavin Young to produce his "Diseases of the Ear, Nose and Throat" for the "Catechism Series" cannot be considered as conferring a benefit on the literature of this specialty.[2] There is no new thought expressed in its pages and being arranged in catechismal form, the information therein is of a necessity disjointed. This purposeless mixing of ætiology, symptomatology, complications *et cetera* of the various diseases makes the work useless as a book of reference to any practitioner requiring an exact knowledge. Its perusal might possibly enable a student to gain a temporary and very limited smattering of information on the subject. The latter appears to be the only excuse for its publication.

[1] "The Mothercraft Manual or the Expectant and Nursing Mother and Baby's First Two Years," by Mabel Liddiard, with an Introduction by J. S. Fairbairn, M.A., B.M., B.Ch. (Oxon.), F.R.C.P., F.R.C.S.; Fourth Edition; 1925. London: J. and A. Churchill; Sydney: Angus and Robertson, Limited. Crown 8vo., pp. 184, with illustrations. Price: 4s. 6d. net.

[2] "Catechism Series: Diseases of the Ear, Nose and Throat," by Gavin Young, M.C., M.B., Ch.B., F.R.C.P.S.; 1926. Edinburgh: E. and S. Livingstone. Crown 8vo., pp. 67. Price: 1s. 6d. net.

The Medical Journal of Australia

SATURDAY, MAY 22, 1926.

The Profession and the State.

EARLY in January the Royal Commission on Health issued its report. On February 3 and 4, 1926, the Federal Committee of the British Medical Association in Australia had the report before it and referred the matter to the Branches. The text of the report was published in THE MEDICAL JOURNAL OF AUSTRALIA on January 16, 1926, and during the subsequent weeks various chapters of the report have been discussed in these columns. It must be evident to every student of hygiene that the problems examined by the Commissioners are of paramount importance to Australia and that since the public health is at stake, there is extreme urgency in the adoption of the recommendations. Of the five Commissioners four are members of the British Medical Association and three have been for many years members of the Federal Committee. Sir George Syme, the Chairman of the Royal Commission and Chairman of the Federal Committee, has stated that he and his colleagues have benefited very greatly by the fact that the Federal Committee had investigated many of the problems included in the terms of reference of the Royal Commission. It does not necessarily follow that the whole profession will accept the recommendations, although it is unlikely that there will be serious dissent. In these circumstances it is desirable that the Branches should consider the report and should send to the Federal Committee comments and an intimation whether they endorse or disapprove of the conclusions and recommendations of the report. It is to be regretted that the matter has not been treated as one of urgency. Weeks and months have passed; the members of the Branches of the British Medical Association in Australia have not manifested any real interest in the matter. The Councils of the Branches do not appear to have recognized the wisdom of devoting time and attention to the numerous problems involved, with the result that enthusiasm has not been awakened among the members. The Federal Committee has requested the Federal Government to grant it an opportunity of examining any measures which may be proposed as a result of the report, before these measures are finally presented to Parliament or introduced under existing statutes. This request indicates that the Federal Committee would be prepared to discuss any proposals without delay or hesitation. If the proposals involve the rank and file of the medical profession, it would be essential to have the assurance of the profession as a whole that its members are in fact prepared to accept the new responsibilities and new duties that may be imposed on them. It is essential that the events of 1911 in connexion with the national health insurance scheme in Great Britain should not be repeated in Australia. In England the medical profession took no interest in the problem of national health insurance until the bill was before Parliament. It was then too late to influence the legislature to modify the proposals, so that the support of the medical profession as a body could have been assured. Much of the opposition to the *National Health Insurance Bill* of 1911 was due to a lack of understanding of the proposals and a failure to grasp the objectives of those responsible for the measure. Had the medical profession been induced to anticipate the introducton of the panel system and to have examined the matter with care and understanding, the unseemly scenes at certain great medical gatherings could have been avoided.

While the whole report is important and urgent, it appears to us that two or three chapters demand immediate consideration. These are the model scheme for the cooperation of the Commonwealth with the States in the measures aiming at the prevention of disease, the institution of an efficient school of hygiene, preventive medicine and tropical medicine at the Sydney University and the transfer of the powers regarding medical registration from the States to the Commonwealth. The model scheme includes the enlistment of the whole medical profession in the campaign. To a large extent the suggestions embodied in the Hone-Newland report have been accepted by the Commissioners. The general practitioner is thus faced with an entirely

new prospect. He will be expected to devote a proportion of his time and energies to preventive medicine; it will become his duty to assist the health authorities to trace the sources of infection and to render them harmless. He will have responsibilities to the State as well as to his patients. He will have to make up his mind whether his services to the State will be based on a bargain, a contract whose clauses are to be translated literally or whether he will be prepared to lend his aid freely for the benefit of the community and pit a reasonable remuneration on the credit side of his sheet against services rendered generally to the State and privileges received from the law and the community in virtue of his position as a registered medical practitioner. A thorough investigation of the report, a further study of the Hone-Newland report and the exercise of a little imagination will give him the key to the situation. He will be able to anticipate the proposals that will form the policy of the Commonwealth health authority and on the basis of what he anticipates, he will be able to decide what he will do. If this is not undertaken quickly, we shall have the proposals thrust forward before the medical profession has made up its mind. It would be a disaster.

Current Comment.

PRIMARY CARCINOMA OF THE LIVER.

PRIMARY carcinoma of the liver is not of very rare occurrence. It has been reported as occurring in children, but more often adults past the age of forty are affected. It is found more frequently in males than in females and it has been pointed out that this sex incidence runs parallel with that of cirrhosis of the liver. Thus in thirty-two cases reported in 1894 by von Henkelom thirty-one of the patients were males. The clinical picture as far as adults are concerned is as a rule typical. The patient may have complained for a considerable time of symptoms such as those generally associated with cirrhosis. Then a sudden exacerbation occurs and the patient rapidly goes down hill and dies within a few months. Sometimes the sudden and severe symptoms leading to death are the first indication of illness.

In considering carcinoma of the liver it is well to remember Virchow's dictum that before a carcinoma can be regarded as primary, it is necessary to make a most exhaustive search for a primary focus in other organs. Tumours have frequently been reported as primary when careful search has shown that the diagnosis could not be substantiated. In a recent study of carcinoma of the liver Dr. V. S. Counseller and Dr. A. H. McIndoe refer to a case in which a huge nodular tumour of the liver was regarded as primary.[1] Subsequent search was undertaken on account of the absence of cirrhosis and the presence of a clear celled structure in the tumour. A nodule 1·5 centimetre in diameter was found in the suprarenal capsule and this was proved to be a "hypernephroma."

The chief point of interest in primary carcinoma of the liver lies in its relationship to cirrhosis of that organ. On a previous occasion the histological changes found in the cirrhotic liver have been discussed.[2] The liver possesses remarkable regenerative power as was first shown experimentally by Ponfick. With the destruction of liver cells in cirrhosis and their conversion into fibrous tissue concurrent efforts at regeneration are manifest. Efforts at regeneration have been noted in so acutely destructive a condition as acute yellow atrophy. It is of interest to note in passing that Epplen regards acute yellow atrophy and cirrhosis of the liver as analogous conditions, differing only slightly in the nature of their pathological processes, but widely in their degree. He regards one as acute and the other as chronic. Adenoma of the liver may be single or multiple. Multiple adenomata are usually associated with cirrhosis and it has been pointed out that this is probably because cirrhosis is the commonest disease which destroys liver cells and makes compensatory hypertrophy necessary. Kelsch and Kiener in 1876 advanced the view that cirrhosis and adenomata are caused by the same poison and Brisand described multiple adenomata as the halfway house between cirrhosis and primary carcinoma. According to Muir no sharp division exists between multiple adenomata without cirrhosis, multiple adenomata with cirrhosis and carcinomata. Each of these conditions in his opinion exhibits progressive, invasive and malignant tendencies. It may be of interest to point out here that Ewing in dealing with primary epithelial tumours of the liver divides them into the following groups: (i.) Solitary adenomata, (ii.) primary massive liver-cell carcinomata, (iii.) multiple liver cell carcinomata and (iv.) carcinomatous cirrhosis.

In the study by Dr. Counseller and Dr. McIndoe to which reference is made above, they report in detail the histories and *post mortem* findings in five cases of primary carcinoma of the liver. The five cases were observed in 5,976 autopsies, an incidence of 0·08%. During the same period 220 cases of secondary carcinoma of the liver and 127 cases of portal cirrhosis were observed. In all five cases a cirrhosis of the portal type was found and in three instances the history pointed to the presence of a preceding cirrhosis. In four the condition was a primary carcinoma of the liver cells and in the

[1] *Archives of Internal Medicine*, March 15, 1926.
[2] THE MEDICAL JOURNAL OF AUSTRALIA, September 2, 1922.

remaining case the tumour was a cholangioma or carcinoma of the bile ducts. In every instance it was possible to decide which was a malignant and which a normal cell and no true transitional forms were found. Hence Dr. Counseller and Dr. McIndoe agree with Winternitz, Karsner and others that the condition is unicentric in origin and that its spread in the liver is by metastasis in the portal and hepatic veins. In only two of the five cases were extra hepatic metastases found. In one case there was an extension through the hepatic veins into the inferior *vena cava,* right auricle and pulmonary artery with small nodules in the lungs. In the second case there was an extension to the spine at the level of the sixth dorsal vertebra. The body of this vertebra was replaced by a soft greyish-pink mass which had extended into the vertebral canal on either side of the cord, causing compression myelitis. This form of metastasis is unusual and Dr. Counseller and Dr. McIndoe can find no parallel to it.

The question as to whether the cirrhosis is primary, secondary or coincident with the carcinoma is discussed by Dr. Counseller and Dr. McIndoe. The view that the two conditions are coincident is in their opinion untenable on account of the regularity of the appearance of cirrhosis and also its generalized and severe form. They refer to the view held by Hanot and Gilbert as to the coincidence of the conditions on the ground that tumour and cirrhosis are produced by the same ætiological agent. They point out that from an analogy with other cases of carcinoma in which chronic inflammatory conditions are almost certainly ætiological, such a view would be difficult to maintain, particularly in regard to malignant change undoubtedly preceded by cirrhosis. In support of the view that a malignant tumour induces cirrhosis even less can be said. Any form of growth will cause localized fibrosis, but never the generalized and severe form seen in this condition. Dr. Counseller and Dr. McIndoe are in full accord with the view that the cirrhosis is the primary factor and that the malignancy is a change superimposed on it. They refer to powers of regeneration possessed by the liver. In cirrhosis there is an actve destruction and an equally active regeneration with an adenomatous condition of the liver cells most pronounced in the portal type. In the connective tissue are bile canaliculi and these have been held to have the power of producing liver cells. Muir denies this and Dr. Counseller and Dr. McIndoe are of the same opinion. They hold that, whatever may be the factors producing the cirrhosis, the path of entry is either the portal or biliary channels. The portal entry is the more common. In a mild case destruction of cells takes place in the neighbourhood of the portal spaces and new connective tissue is laid down. In order that this process may continue, two factors are necessary, cell death and the continuous action of a toxin. The rapidity of the process and its extent will be modified by the degree of regeneration of the liver cells and bile ducts. In the portal type the toxic agent affects primarily the cells in the neighbourhood of the portal spaces. In the biliary type the toxic agent being more confined to the bases of Sabourin's acini, the so-called monolobular fibrosis results. At the same time Dr. Counseller and Dr. McIndoe emphasize the fact that in neither type is an anatomical basis for classification justifiable, for the process is governed by the resultant of the factors already mentioned and not by the influence of the individual lobule. As biliary cirrhosis is more uncommon and less severe than portal cirrhosis, regeneration is not such a prominent feature and hence is rarely seen in conjunction with carcinoma.

In conclusion these observers refer to the view that adenoma, nodular hyperplasia and carcinoma "insensibly shade off one into the other." Such an occurrence in their opinion would be easy to understand were not carcinoma so extremely rare when compared with cirrhosis and regenerative changes in the liver. Some specific factor or specific change in environment is responsible for the transition from hyperplasia to a malignant condition of the cells, something which "removes the barrier confining their biologic function of growth and regeneration within normal limits." They are of the opinion that from 3% to 4% of cases of cirrhosis will develope into carcinoma "as the direct result of the changes induced by the chronic inflammatory condition." Presumably then the chronic inflammatory condition supplies the alleged specific factor.

POLYPOSIS OF THE COLON FOLLOWING ULCERATIVE COLITIS.

VERSE held the view that the sole element of predisposition in the causation of polyposis of the colon was the excessive reaction of the epithelium to irritants. In THE MEDICAL JOURNAL OF AUSTRALIA of June 13, 1925, reference was made to the views of Erdmann and Morris that in polyposis of the colon the sequence of irritation, inflammation, reaction, mucous membrane hyperplasia and polypus formation was readily acceptable.

Mr. J. G. Kingsbury has recently reported a case of polyposis which possibly lends some support to this view.[1] A man, aged forty-six years, was admitted to hospital suffering from ulcerative colitis. The ulcers were seen on sigmoidoscopic examination. Treatment was carried out with antidysenteric serum and an autogenous vaccine prepared from Morgan's Number 1 bacillus which had been isolated from the stools. With the improvement of the colitis small sessile polypi appeared and were found scattered over the whole area accessible to the sigmoidoscope. Four months later a barium enema was given and small translucent areas could be recognized in the descending colon. It was thought that these might have been due to polypi larger than those in the pelvic colon. Mr. Kingsbury states that as far as he can ascertain this is the first recorded case of multiple polypi occurring as a sequel to ulcerative colitis.

It is interesting to note that the polypi disappeared as a result of deep X ray therapy.

[1] *Guy's Hospital Reports*, January, 1926.

Abstracts from Current Medical Literature.

MEDICINE.

Pulmonary Tuberculosis in Childhood.

L. FINDLAY (*Edinburgh Medical Journal*, March, 1926) discusses pulmonary tuberculosis and allied conditions in infancy and childhood. He expressly excludes discussion of changes in the mediastinal glands. In infancy and childhood pulmonary tuberculosis is usually of the acute broncho-pneumonic type and there is usually a generalized infection of the lungs. This condition occurs in Glasgow most frequently in the first three years of life. Chronic pulmonary tuberculosis is a very rare condition in childhood. Unresolved pneumonia with bronchiectasis is often mistaken for tuberculosis. Pulmonary tuberculosis in childhood is generally widespread in both lungs. The radiologist has been too prone to diagnose pulmonary tuberculosis. Findlay can recall only one questionable case of chronic pulmonary tuberculosis among all the *post mortem* examinations carried out at the Children's Hospital in Glasgow and he has never seen calcareous glands in the mediastinum, though they are often diagnosed by X ray examination. The presence of an enlarged spleen and enlarged glands is an aid to diagnosis of tuberculosis. Sputum may be obtained by swabbing a child's throat with the finger guarded by a bit of gauze, coughing is induced and sputum voided on to the gauze. A reaction occurs so often to the von Pirquet test that it is not of much value.

Nephritis in Children.

S. W. CLAUSEN (*Atlantic Medical Journal*, January, 1926) discusses the causation and treatment of acute nephritis in children. Two varieties are recognized. The first is glomerular nephritis due to streptococci usually and associated with hæmorrhages, anæmia, hæmaturia, nitrogen retention, raised blood pressure and possibly retinitis, death being due to uræmia. The second variety is tubular nephritis (parenchymatous) with definite œdema and none of the other signs mentioned above, death occurring from intercurrent infection. Tonsillar infections, chronic upper respiratory infections and many infectious diseases precede glomerular nephritis as a rule, the capillaries manifesting widespread injury throughout, spasm and tortuosity being observed even at the base of the finger nail. Treatment includes restriction on some days to sugar only, ten grammes of cane sugar per kilogram of body weight being dissolved in one and a half litres of fruit juice and drunk freely. This often benefits hæmaturia. In severe cases sweating and purgation are not employed; intravenous injections of 20% glucose sometimes lowers blood pressure and causes diuresis. Intravenous injection of magnesium sulphate 0·2 gramme per kilogram of body weight in 2% solution was of benefit to other patients in a similar way and occasionally relieved painful cramps in uræmia. Sweating is also promoted in some cases by means of sugar or magnesium sulphate given intravenously. Tubular nephritis is often associated with nasal sinusitis and sometimes with other infections. Treatment of the infection is necessary, salt is limited in the diet which contains three grammes of protein per kilogram of body weight. Diuretin and intravenous injection of sugar (glucose) or magnesium sulphate are useful in causing diuresis and in mental states which are attributed to cerebral œdema. In both conditions treatment of any focus of infection is essential.

Hilum Tuberculosis.

J. B. HAWES (*Boston Medical and Surgical Journal*, January 21, 1926) discusses the diagnosis of clinical hilum tuberculosis. The following points are suggestive: a definite history of exposure, severe whooping cough or measles followed by debility, enlarged cervical glands and a history that the child has been delicate. Unusual fatigue and change of temperament are common symptoms of hilum tuberculosis; cough and sputum, sweats, chest pains, fever and rapid pulse are not very useful in differentiating the disease from others. The chest examination clinically is of little value in early diagnosis as a rule. The X ray picture is useful, but a diagnosis of active or inactive tuberculosis cannot be made by means of X rays. Real malnutrition is a valuable sign. With careful interpretation von Pirquet's test has a useful field.

Treatment of Sciatica.

E. STRÄUSSLER (*Wiener Medizinische Wochenschrift*, January 23, 1926) considers that in acute cases of sciatica the first line of treatment is the application of dry heat to the affected leg. Quinine (one gramme) is then given and repeated in a dose of half a gramme. The following day the latter dose is repeated combined with "Aspirin" (two grammes at least), while the patient is given hot drinks and placed in a pack. According to the success achieved this treatment is kept up for fourteen days, though other antirheumatic drugs may be used instead of quinine. It is important that the treatment be maintained and not stopped because of improvement. With these methods recovery will occur in many acute cases. For intractible and chronic cases gentle stretching of the nerve while the patient is in a hot bath combined with massage is frequently necessary. In addition electrical treatment, either galvanic or high frequency current, may be found useful. Injection of the nerve with seventy to one hundred cubic centimetres of a 0·1% β eucaine solution causes rapid relief of pain. It is frequently followed by a rise in temperature and even a rigor so that rest in bed for several days is essential. This resorption fever may be obviated by adding calcium chloride 0·75 gramme to a litre of the saline solution. Good results have also been noted following the use of a 50% solution of antipyrin with or without 1% "Novocaine." If the lesion be suspected to exist in the terminal filaments of the spinal cord, epidural injections of normal saline solution may be of great service. Apart from these methods the author has tried injections of "Vaccineurin" with great success. This treatment follows the line of the modern protein therapy. For resistant cases it should be combined with pyocyaneus vaccine.

Prophylactic Blood Transfusion.

G. F. WARD (*Illinois Medical Journal*, February, 1926) considers that a very definite improvement in mortality and morbidity statistics would follow routine blood transfusions prior to serious operative procedures. All hospital internes should, he says, be familiar with the technique of blood-typing. Five hundred cubic centimetres of blood are usually sufficient for prophylactic purposes and will produce no distress in the donor. Many surgeons do not appreciate the amount of blood lost during operations. The case is cited of a panhysterectomy in which three hundred and four cubic centimetres of blood ran away unheeded and of an operation for nephrectomy in which the blood loss was eight hundred and sixteen cubic centimetres. In a patient with a hæmoglobin value of 50% or less or with less than three million erythrocytes per cubic centimetre of blood such sacrifices may well prove fatal.

Sea Sickness.

P. H. DESNOES (*Journal of the American Medical Association*, January 30, 1926) has devoted attention to the subject of sea sickness. He considers it due primarily to excess stimulation of the labyrinth. Eye strain, however, as well as disturbance of the muscle sense, peripheral stimulation of the vagus nerve and psychical impressions are additional factors in the production of the malady. In treating the condition the author reports good results from the use of scopolamine hydrobromide in small doses, 0·00015 gramme (one four hundredth of a grain), administered by the mouth and repeated at hourly intervals until the patient obtained relief. Any depression due to the sickness may be combated by combining strychnine in doses of 0·001 gramme (one sixtieth of a grain) with the scopolamine. Psychical disturbances may be overcome by some centrally acting hypnotic such as chlorbutanol in doses of 0·3 to 0·6 gramme (five to ten grains). Very excessive vomiting sometimes requires the use of morphine hypodermically and that the patient be fed whilst remaining prone with such restoratives as iced brandy or champagne and strong coffee. Desnoes states that the hydrogen ion concen-

tration of the blood must be reduced and an attempt made to store up "buffer" substances in the blood to counteract the tendency to acidosis. Such salts, therefore, as are of acid reaction (for example those of meats, cereals and legumes) may be fitly replaced by the salts of tubers, fleshy roots, leafy green vegetables and citrus fruits in order to increase the alkali reserve of the blood.

Hepatic Functional Tests.

U. FRANKE (*Klinische Wochenschrift*, January 1, 1926) after describing the tetrachlorphenolphthalein test for hepatic function discusses the results he obtained in thirty cases. In some of these when the liver was apparently normal, the nature of the response was doubtful. He considers that a colour reaction obtained with a 3% soda solution means nothing; if a 5% solution be required, then the satisfactory nature of the liver function is doubtful. When an 8% soluton is necessary there is definite evidence of hepatic insufficiency. The use of the test is not without some serious after effects. Many patients suffer from headaches, fainting and lassitude, while others manifest a severe local reaction round the site of injection. The author considers that the results so far obtained justify further investigation.

Focal Infections.

S. E. EASON (*New Orleans Medical and Surgical* Journal, February, 1926) contributes a *résumé* on the subject of focal infections. Up to the present Rosenow has shown that such lesions as peptic and duodenal ulcers, endocarditis, appendicitis and many other serious lesions may be due to foci of infection in the teeth, tonsils and lymph glands. Bacteria from the infected teeth of a person suffering from myositis, for example, have been injected into the veins of a rabbit. The rabbit, when killed, has been shown to be suffering from hæmorrhage and degeneration in muscles identical morphologically with those of the original patient. Cultures from these affected muscles, furthermore, will produce the same pathological results as cultures taken from the original dental focus. The bacteria taken from the tonsils of a sufferer from rheumatism, even though the tonsils to outward appearance were only slightly diseased, have produced the lesions of rheumatism in a rabbit, but cultures taken from tonsils apparently more diseased, the patient being free from constitutional evidences of rheumatism, have produced no arthritic changes in animals. Infective foci, therefore, are of very variable virulence. Probably the most virulent organism is the *Streptococcus hæmolyticus*, but many other germs (*Bacillus tuberculosis* pneumococci, streptococci of other varieties) are serious offenders. Curtis, of Chicago, has described the case of a woman who aborted within one year after the birth of a sickly, undersized child. The woman's urine on culture was found to contain *Streptococcus hæmolyticus*. Intravenous injection of the culture into pregnant rabbits caused either abortion or absorption of the fœtus in every experiment. It was discovered later that the woman's husband, a diabetic, was the victim of dental and urinary infections, from both of which the *Streptococcus hæmolyticus* was grown in culture. The removal of his infected teeth caused the signs and symptoms of diabetes to disappear, while the woman on her part gave birth in due course to a healthy child.

Renal Function in Scarlet Fever.

G. H. PERCIVAL AND C. P. STEWART (*Edinburgh Medical Journal*, February, 1926) have recently been engaged in the determination of the urea content of the blood as a test of renal function during the course of scarlet fever. All the patients, thirty-six in number, were fed on milk or farinaceous foods up to the twenty-first day of the disease; thereafter fish or white meats were added. In every instance care was taken during the first ten days of illness to exclude the possibility of a preexisting nephritis. The research shows that during the initial pyrexial stage of scarlet fever there is a tendency to a high blood urea content, but no connexion can be seen between the urea content and the severity of the pyrexia. The high blood urea content, however, is temporary only and is succeeded by a drop to the normal level. After the tenth day or thereabouts a second rise of blood urea occurs whether or not clinical signs of nephritis are present. The important point, however, is that the fall to normal is rapid in non-nephritic patients, very much slower in those who become affected by nephritic changes. The authors also observe that even when there are present all the cardinal signs of nephritis (oliguria, hæmaturia, albuminuria, œdema *et cetera*) the blood urea may still be low. The conclusion to be drawn is that no warning of impending nephritis can be obtained by determinations of urea in the blood.

Excretion of Alcohol in Urine as a Guide to Alcoholic Intoxication.

GODFREY CARTER (*The Lancet*, January 23, 1926) contends that all the present tests for inebriety are inadequate. No two men in poise, gait, powers of memory will react alike to the influence of alcohol and the breath smells as strongly after one drink as after many. Carter regards as important signs the flushing of the face, sweating, dilated pupils, congested eyes and a rapid pulse. The determination of drunkenness has risen into prominence owing to the charges preferred against motor drivers. The work of Southgate shows that the curve of alcohol concentration in the blood after the administration of a known quantity of alcohol is remarkably definite and constant. The curve of the alcohol excreted in the urine is of similar nature. At any moment after alcohol is taken it is possible by examination of the urine to estimate with some accuracy the amount of alcohol in the blood and thus to say whether this concentration is enough to produce inebriety. Carter who is police surgeon, has employed this method upon twenty-five persons arrested for drunkenness and is satisfied as to its value. Southgate states that the concentration in the blood is proportionate to the toxic effect produced and that as above stated the concentration in the blood can be deduced from that in the urine. The concenération curve of the blood alcohol rises rapidly to a maximum in about one hour and falls slowly to reach zero in about twelve hours. Thus the curve of disappearance is a practically straight line. A blood glucose curve rises with equal rapidity but reaches normal, eighty to one hundred milligrammes, in about one and a half hours. The kidney which keeps back glucose in a high percentage, has not this power over alcohol. Even a half-glass of beer will cause excretion of alcohol. The actual maximum concentration varies with the dose taken and rises much more slowly when the alcohol is mixed with food.

Relation of the Vagus Nerve to Gastric and Duodenal Ulcer.

O. SCHMIDT (*Wiener Medizinische Wochenschrift*, August 15, 1925) states that as a result of his experiments he can see no truth in the neurogenic theory of the causation of gastric and duodenal ulcers. He has never seen gastric ulceration follow damage to the vagus nerve or myelitis. Similarly in cases of ulcer he has not noticed any histological changes in the nerve.

Treatment of Pneumonia.

J. C. MEAKINS (*The Canadian Medical Association Journal*, January, 1926) discusses the modern treatment of pneumonia. The results of treatment with Type No. 1 pneumococcus antiserum are encouraging when the type of organism is determined and the specific antiserum is used early in the disease. Elimination of toxins by bowels and kidneys is necessary, the latter being especially effected by copious internal hydrotherapy. Dyspnœa is best combated by the semirecumbent posture and the early use of oxygen, administered by catheter through the nose or mouth or by a close fitting mask. Of respiratory stimulants carbon dioxide is the one which produced greatest increase in depth of respiration, but its therapeutic use is not yet satisfactory. Exposure of the face to cool air causes respiratory stimulation and fresh air may act in this way. Digitalis can be used early in the disease, especially as a cardiac stimulant and to prevent cardiac dilatation. Efficiently given oxygen is valuable in the treatment of myocardial failure and of nervous and circulatory symptoms. Delirium can be prevented or removed by the elimination of oxygen want. The air of the sick room should circulate freely, if necessary aided by fans.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Lister Hall, Hindmarsh Square, Adelaide, on March 25, 1926, DR. C. T. C. DE CRESPIGNY, D.S.O., the President, in the chair.

Peptic Ulcer.

DR. C. F. PITCHER read a paper entitled: "Some Observations on Peptic Ulceration: With Special Reference to its Medical Management" (see page 569).

Radiology and Duodenal Ulcer.

DR. H. A. MCCOY read a paper entitled: "The X Ray Diagnosis of Duodenal Ulcer" (see page 574).

DR. A. CUDMORE thanked Dr. Pitcher for his paper. He was of the opinion that gastro-enterostomy cured the majority of patients with chronic peptic ulcer.

DR. B. SMEATON thought that the majority of patients with peptic ulcer were cured by gastro-enterostomy, in a minority failure occurred and the failure was either complete or only temporary relief was afforded. He outlined his procedure with his patients at the Adelaide Hospital. They were required to report regularly after operation. Their symptoms were classified and usually a fractional test meal was done for comparison with that done before the operation.

DR. H. S. NEWLAND, D.S.O., laid stress on the use of medical after treatment as regards diet and administration of alkalis in the period following gastro-enterostomy. The value of gastro-enterostomy in gastric ulcer, he stated, was much less, except in cases of pyloric ulcer.

In cases of ruptured ulcer where no gastro-enterostomy had been done, patients were sometimes apparently cured possibly because of the careful dieting. He, however, advocated a gastro-enterostomy to be performed at the first operation, if the patient could stand it.

DR. J. CORBIN agreed with the value of gastro-enterostomy in the treatment of chronic peptic ulcer. He also produced the results in a series of patients with ruptured ulcer which had been treated by suture and invagination of the ulcer or with gastro-enterostomy in addition. He suggested invagination of the ulcer combined with gastro-enterostomy as a more certain cure of an unperforated peptic ulcer.

DR. F. S. HONE supported Dr. Pitcher's remarks that many patients with gastric ulcer could apparently be as successfully treated by medical treatment and dieting. He considered that prolonged careful dieting was essential after gastro-enterostomy and urged the need for close cooperation between the physician and surgeon in studying the after history of these patients.

DR. J. VERCO referred to some of his patients whose symptoms had been relieved by the administration of belladonna. The rationale of this treatment was the relief of pyloric spasm.

PROFESSOR J. B. CLELAND stated that gastric and duodenal ulcers appeared to be unduly common in the insane. During the previous few years nearly one hundred autopsies had been carried out at the Parkside Mental Hospital and in four cases either gastric ulcers or necrosed patches about to become ulcers were present. There were also five instances in which duodenal ulcers were detected. Two of the cases of gastric ulcer were of more especial interest as showing possible antecedent causes for the lesions. Thus a male, *ætatis* forty-eight years, had mitral stenosis, an *ante mortem* clot in the left auricular appendage, infarcts in the right lung and no less than four subacute gastric ulcers with a necrosed patch in addition which was obviously about to become an ulcer. In this case it was possible that infarcts were responsible for the development of the ulcers.

The second case was a male, *ætatis* thirty-eight years, an imbecile, with necrotic patches in the stomach about to become ulcers. The patient had subcutaneous abscesses, pyæmic foci in the lungs, pyelitis and abscesses in the kidneys and a superficial bedsore, ample sources of infection if infection was sometimes the cause of gastric ulcer.

Odontome.

PROFESSOR J. BURTON CLELAND described and showed a large odontome (see page 580).

University Intelligence.

THE UNIVERSITY OF MELBOURNE.

AFTER a period of twelve years the Government of Victoria has given its sanction to an essential reform in connexion with the Medical School of the University of Melbourne. It will be within the recollection of the readers of THE MEDICAL JOURNAL OF AUSTRALIA that on October 19, 1914, the Council of the University considered a scheme formulated by Professor R. J. A. Berry for the rebuilding of the Medical School on a site adjoining the Melbourne Hospital. The proposal had wide support both within the medical profession and in other circles. It appealed at once to the University on account of substantial advantages that would have accrued to the Faculties of Science, Agriculture, Law, Arts and Medicine. Had the Medical School been rebuilt in Lonsdale Street, the old buildings would have become available for the other faculties. The Government had promised to expend some £80,000 on new buildings for the Faculty of Arts and for the administrative offices of the University. Professor Berry asked for an additional sum of £120,000 which with the amount already promised would have sufficed to have doubled the accommodation in five faculties. The proposals were not adopted. In the interval large sums of money have been expended both in the University and in the Melbourne Hospital for building operations. It is not suggested that too much has been erected. Far from it. The University has been supplied with but its minimum requirements. Soon after the refusal by the Government to adopt the plan the site was purchased for another purpose and with this Professor Berry's original scheme had to be abandoned.

More recently other proposals have been made to effect the same reform. If the Medical School could not be moved to the largest and most modern general public hospital, there remained the alternative of erecting a clinical hospital near the Medical School. Difficulties presented themselves in the elaboration of this plan. The existing Medical School does not lend itself well to an intimate linking up with a new hospital on any available site.

It must be pointed out that Professor Berry's original proposal involved the erection of a structure of six storeys. To bring the laboratories and class rooms within easy reach of the clinic Professor Berry was prepared to climb up into the sky. In medical schools of what may be described as the purely clinical type the fundamental sciences are taught in cramped surroundings in close proximity to the hospital. In the other extreme type the academic studies are given first consideration, while the clinical arrangements are haphazard and inconvenient. The modern conception of medical education demands an adjustment between scientific study and clinical practice. It is regarded as essential that the student should realize the application of the chemical, physical and biological principles which he masters in the laboratory, to the problems of disease. The class rooms, laboratories, lecture theatres and libraries should be commodious, well equipped, properly constructed and removed from disturbing influences. At the same time the hospital should be within easy reach of these homes of scientific training and should contain subsidiary laboratories, research rooms, lecture theatres and reading rooms. We have maintained for many years that the medical student should continue to study the fundamental sciences throughout the entire curriculum. If land be available and its value not very high, the medical school is best spread over a considerable

area, provided that the student is not required to lose valuable time in passing between it and the hospital.

The New Scheme.

Toward the end of 1922 the hospitals of Victoria were placed under the control of the Charities Board under the *Hospitals and Charities Act* and since then a system has been created which places these institutions among the best organized hospitals in the world. The Charities Board has evinced a great interest in the problem on the solution of which Professor Berry has worked so arduously and patiently. In 1917 Parliament sanctioned the purchase of the Pig Market site, an area of land practically adjoining the University lands. For several years the Department of Education has endeavoured to secure the use of this site. The Council of the University also cast a longing glance in the same direction. After a considerable amount of finessing, the Faculty of Medicine made the proposal that the site should be used for the erection of a new medical school with a clinical hospital. The proposal was strongly opposed by the Department of Education. The Charities Board took a hand in the arguments and with the Faculty of Medicine induced the Government to appoint a committee to inquire into the claims of all interested parties. The committee accepted the charge and conceived that the best way of arriving at a sound and wise conclusion would be to invite the Council of the University, the Department of Education, the Charities Board, the Committee of the Melbourne Hospital and the Faculty of Veterinary Science to send delegates to confer with it. The composition of the conference will disarm criticism; the public interest has certainly been safeguarded. This conference submitted a report to the Premier of the State and on April 23, 1926, the Government agreed to give effect to the recommendations.

The signatories of the report state that at the outset of the discussions it was understood that if the proposals made for the purpose of satisfying the educational needs involved an appeal to Parliament to make available lands reserved for other purposes and Parliament declined to accede to the request, the whole position would have to be reviewed. It was further agreed that the Charities Board and the Medical School should accept their apportionments on condition that the interests of the other bodies represented in the conference were conserved. The conclusions were adopted by the unanimous vote of the members. It was realized that none of the bodies represented would obtain all it asked for. On the other hand the land and buildings which each would receive, if the agreement were adopted, would suffice for its immediate needs and the whole scheme would be of the greatest value to the community for many years. As far as the Faculty of Medicine is concerned, it would seem that what is offered, will solve the present difficulties and will enable it to adopt satisfactory reforms in the near future.

The University High School.

The Department of Education is to have a block of land of about five and a quarter acres, known as the Old Carlton Cricket Ground for the University High School. This is to serve as a district high school and a practising school for the training of the secondary school teachers of the State. An area of eight acres of the Pig and Cattle Market site is to be provided for an enlarged teachers' college and residential accommodation for the students at the teachers' college. Lastly there is to be a block for an hostel for students of domestic arts.

A College for Women Students.

The University is to receive an area of three and a half acres facing Madeline Street to be used for the University Women's College.

The New Hospital.

The Charities Board is to have an area of eleven and a half acres of land including the Hay Market and portion of the Pig and Cattle Market for a teaching hospital. It is suggested that the Melbourne Hospital will be transferred by gradual stages to the new site. It may be assumed that the conference held the opinion that this transfer should be a very slow process. It is pointed out that the metropolis of Melbourne is very badly off in regard to hospital beds, as compared with the other capital cities of Australia. While Brisbane has 4·3 beds per thousand of population, Sydney has 3·7 and Perth has 3·3, Adelaide has only 2·0 and Melbourne 1·9 beds per thousand of population. The policy of the Charities Board to establish district hospitals at suitable situations with sufficient accommodation to meet the needs of the community necessitates the erection of a large hospital in the Carlton district, to the north of the city. It is proposed that the Melbourne Hospital will be handed over to the Government as the buildings are released from service. The site is of great value and the buildings which are practically new, will have a considerable pecuniary worth. As the recommendations include the accommodation in the Medical School and general hospital of the Dental College and Dental Hospital, the existing Dental College will be sold and the proceeds will be added to the funds needed for carrying out the complete scheme.

The Medical School.

In the last place the Medical Faculty has been given a site of about two acres for the purpose of a new Medical School. This site is at the corner of Sydney Road and Grattan Street and thus close to the spot where the new hospital will be erected. In addition the Medical Faculty will have control of a clinical unit with the hospital.

We understand that the building favoured will be one constructed largely of reinforced concrete with an architectural style similar to that of the Molteno Institute of Parasitology at Cambridge. The frontage to Grattan Street will measure two hundred and forty-four feet, while the depth will measure two hundred and twenty-five feet. There will be a central courtyard with four sides or wings. The buildings will be three storeys high with an entresol and towers. In other words, there will be a ground floor, an entresol, a first storey and a second storey and towers on the main buildings. The hospital is to the west of the school. It has therefore been determined to place the departments of pathology and bacteriology in the western wings. The administrative apartments will be in the southern part or main building, while in the eastern wing the departments of physiology and anatomy will be housed. Adjoining the pathological section will be the great Museum of Pathology which owes its extraordinary didactic value to the genius of Harry Allen. Between this and the department of anatomy will be the Anatomy Museum. The ground floor will also contain the department of operative surgery, tucked away to the north-eastern corner, so as to bring it in close proximity with the department of anatomy and at the same time to secure for it the necessary isolation.

On the first floor the eastern wing will be occupied by the departments of experimental medicine, experimental physiology and experimental biochemistry, while in the western wing accommodation will be found for the workers in experimental pathology and pathological biochemistry. The greater part of the second floor will be reserved for the larger research laboratories and for the library. The kitchen and refectory will be situated in the north-western tower, while the north-eastern tower will be reserved for the photographic department. On the ground floor and on each of the upper floors will be animal yards facing the central courtyard. The operating rooms for experimental research will also be placed on these verandah-like extensions to the centre.

It is proposed to provide three lecture theatres to accommodate one hundred and fifty students each, one large theatre for three hundred students, a dissecting room for three hundred students and four, five or six teaching laboratories. The staff will have a series of private rooms and research rooms.

It is premature to anticipate further details. Many of these have been considered, but until the final plans of the architect have been approved, many of these details will probably be modified and altered. Sufficient has been said to render it clear that the scheme has been evolved

with care and precision and that in the near future the Medical School in Melbourne will enter on another phase of its existence. Hitherto excellent work has been accomplished in the stately but somewhat unfavourable old University buildings. The School holds a very high position in the academic world, despite the disadvantages of an unmodern scheme of housing. Once the new buildings are erected and occupied, the University of Melbourne will reap all the advantages of modern construction and equipment and the corresponding disadvantage of having to maintain a still higher degree of efficiency than it already possesses. We have no misgivings concerning the future of the School; the men and women responsible for the teaching and research work will have no difficulty in making the best use of their added facilities for good work.

Congress Notes.

THE AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION).

THE Executive Committee of the Second Session of the Australasian Medical Congress (British Medical Association), Dunedin, 1927, has forwarded to us some preliminary information concerning the facilities for travel to and in New Zealand for members proposing to attend the session.

Concessions on Australian Railways.

It is anticipated that the usual concessions will be granted by the several State Governments for travel within the States *en route* to Dunedin. Forms will be furnished by the Honorary Local Secretaries to enrolled members on application when they are available.

Concessions in New Zealand.

Members of Congress and their wives travelling from the several States of Australia will be granted first class accommodation at second class rates. This concession will be available for one month and will apply to the whole of the New Zealand railway system and not simply to the journey to and from Dunedin. A separate concession form will be required for each single or return journey.

Forms for tickets at concession rates for the journey to Dunedin will be supplied by the Honorary Local Secretaries. Forms for subsequent journeys will be obtainable from the Honorary General Secretary, Professor W. P. Gowland, in Dunedin.

Concessions for Steamship Travel.

The Union Steam Ship Company has consented to allow a 10% reduction in fares from Australia to New Zealand for members and their wives attending Congress. The Company has further agreed to send a steamer from Melbourne, *viâ* Bluff. If desired, this steamer will call at Milford Sound. The steamer will return by the same route. Concession forms will be issued through the Honorary Local Secretaries. Members proposing to take advantage of these arrangements are advised to communicate with Dr. F. Brown Craig, 149, Macquarie Street, Sydney, for further information.

Post-Graduate Work.

COURSE IN OBSTETRICS AND GYNÆCOLOGY IN MELBOURNE.

THE MELBOURNE PERMANENT COMMITTEE FOR POST-GRADUATE WORK has arranged with the members of the staff of the Women's Hospital, Carlton, for the holding of a post-graduate course in obstetrics and gynæcology from August 12 to August 25, 1926. The hospital will be thrown open to those enrolled for the course. There will be no medical students at the hospital during this period. Arrangements will be made for graduates to have access to all parts of the hospital during the whole of the period, both during the day time and during the night time.

Arrangements will also be made for eight graduates to reside at the hospital during the fortnight of the course. If the number of those desiring to go into residence at the hospital exceeds the accommodation, it may be necessary to restrict the time allotted to each to one week.

A fee of two guineas will be charged for the course. This fee will entitle the member to attend all demonstrations and to witness the routine work at the hospital during the whole period. A further fee of four guineas per week will be charged to those who take up their residence in the hospital.

Members proposing to attend the course should notify the Honorary Secretaries of the Committee (Dr. J. W. Dunbar Hooper and Dr. Harold Dew). The fee should be enclosed with the application for registration. They should also intimate whether they wish to go into residence at the hospital.

Correspondence.

ULCERATION OF THE LEG.

SIR: I was interested to read the letters on the treatment of chronic ulcer of the leg. I tried many treatments here and found none of them very satisfactory. I now use a better and a very simple treatment. The ulcer is exposed to direct sunlight for an hour and then a simple antiseptic dressing applied. The leg is covered with a wire gauze cage to prevent flies from contaminating the ulcer. In a few days healthy granulations appear and healing goes on quickly.

Yours, etc.,

E. MACKENZIE.

Malo, New Hebrides,
March 15, 1926.

Obituary.

ROLAND MASTAI LANE.

THE death of Dr. Roland Mastai Lane, whose name was for many years associated with a busy practice in Footscray, Victoria, occurred at St. George's Hospital, Kew, on March 14, 1926.

Roland Mastai Lane, only son of the late James Hogan Lane, was born at Braybrook, Victoria, on January 26, 1878. After preliminary education at a private school in South Melbourne and the University High School, he entered the Medical School at the University of Melbourne in 1896. Graduating in 1901, he availed himself of the opportunities for post-gradute training afforded by resident appointments at the Women's Hospital and Children's Hospital, Melbourne, the Ballarat Hospital and Saint Vincent's Hospital, Sydney.

Roland Lane was prominently identified professionally and socially with Footscray, where he conducted an extensive general practice for seventeen years. In 1921 he left Footscray and went to Brighton, but was soon obliged to retire from practice altogether on account of his ill health. His death was preceded by a long illness which he bore with exemplary fortitude.

"Rowley" Lane was noted for his proficiency in athletic pursuits and in his younger days was a well known amateur cyclist. He was one of the founders of the Footscray Football Club, of which body he was a patron and ex-President. As a holiday pursuit fishing was always his first choice.

In the conduct of his practice Roland Lane never departed from the highest principles of medical ethics. His never tiring service to the poor of his district is well known to his friends and associates and bears witness to his largeness of heart and love of his fellow men. To his great

regret, his services were not accepted for military duty abroad during the Great War, but he did not spare himself in carrying out duties assigned to him in various camps in Victoria during the whole period of the war.

Mrs. Lane, *née* Kinke, daughter of G. Wharton Kinke, Esquire, of Manly, New South Wales, and a family of seven children survive him.

WILLIAM HENRY BROWN.

BY the death of Dr. William Henry Brown, of Sorrento, and formerly of Colac, Victoria, which was recorded in our issue of May 15, 1926, a distinguished figure was removed from the ranks of the medical profession in Victoria.

Born at Erith in Kent, sixty-six years ago, William Henry Brown was educated at Mill Hill, England, and Peterzell, Black Forest, Germany. He was an *alumnus* of University College Hospital, London, and at this school of medicine he was a contemporary of the late Victor Horsley and Sir Henry Maudsley, of Melbourne. In 1885 he came to Victoria and established himself first in Maffra, where he remained six years and later in Colac, where he resided and practised until his retirement thirty years later.

William Henry Brown will be remembered not only as a competent surgeon of wide reputation, but also as an original thinker and investigator. He was a frequent contributor to medical journals, his papers being notable for accurate clinical observation and sound deduction. His best known work gained him great distinction and with it his name will always be associated, for he elucidated the relationship of parathyreoid deficiency to tetany by means of successful implantation experiments. He never omitted to keep himself abreast of the times in scientific matters of medical interest and that he successfully carried out such notable research, although occupied with the cares of medical practice, is eloquent testimony to his boundless energy and exceptional ability.

A man of wide culture, William Henry Brown for many years found relaxation in the study of continental literature in the French and German languages. In public matters he was a leader in his district. Imperial politics and the development of the Labour movement were subjects of great interest to him and he personally initiated a "round table" circle for the discussion of questions of national and international importance.

For practically the whole of the period he was in Colac William Henry Brown acted as Health Officer to the Shire of Colac and a long and distinguished record marks his association with the Colac District Hospital as Honorary Medical Officer.

In 1921 he retired from practice and took up his residence in Sorrento. He was elected a member of the Council of the Shire of Flinders and for a year held the office of President. In outdoor recreation he was an enthusiast in tennis, a game in which he excelled and which he continued to play until a short time before his final illness.

He is survived by his widow, two sons and three daughters. The elder son is Dr. A. E. Brown, of Colac.

Proceedings of the Australian Medical Boards.

NEW SOUTH WALES.

THE undermentioned have been registered under the provisions of the *Medical Act*, 1912 and 1915, as duly qualified medical practitioners:

Abbott, George Sydney, M.B., Ch.M., 1926 (Univ. Sydney), 69, Liverpool Road, Summer Hill.

Armstrong, Allan Cameron, M.B., Ch.M., 1926 (Univ. Sydney), Wondalga, Chelmsford Avenue, Epping.

Belisario, John Colquhoun, M.B., Ch.M., 1926 (Univ. Sydney), 1, Manar Flats, Macleay Street, Potts Point.

Biggs, Thomas James, M.B., Ch.M., 1926 (Univ. Sydney), 91, Milson Road, Cremorne.

Boesen, Carl, M.B., Ch.M., 1926 (Univ. Sydney), Mitcham, 3, Darley Road, Randwick.

Buckingham, Reginald Eric, M.B., Ch.M., 1926 (Univ. Sydney, Carnacoo, Brentwood Avenue, Turramurra.

Burne, Alfred Rainald Keith, M.B., Ch.M., 1926 (Univ. Sydney), 6, Parkes Street, Kirribilli.

Byrne, James Michael, M.B., Ch.M., 1926 (Univ. Sydney), Sierra, Holden Street, Ashfield.

Callow, Francis Henry McClements, M.B., Ch.M., 1926 (Univ. Sydney), Harrow Road, Stanmore.

Clayton, George Edward Burdekin, M.B., Ch.M., 1926 (Univ. Sydney), 22, Arundel Street, Forest Lodge.

Colvin, Clifford Stirling, M.B., Ch.M., 1926 (Univ. Sydney), Ontario, Gerald Avenue, Roseville.

Culey, Arthur Charles, M.B., Ch.M., 1926 (Univ. Sydney), Neirbo Avenue, Hurstville.

Darton, Jack Kenneth, M.B., Ch.M., 1926 (Univ. Sydney), Meavy, New Canterbury Road, Petersham.

Davis, Keith Joseph Brandon, M.B., Ch.M., 1926 (Univ. Sydney), 34, Gowrie Avenue, Waverley.

Day, Emily Martha Anning, M.B., Ch.M., 1926 (Univ. Sydney), 5, Bishop's Avenue, Randwick.

Dickson, Ian Thomas, M.B., Ch.M., 1926 (Univ. Sydney), 81, Beach Street, Coogee.

English, William Gerard, M.B., Ch.M., 1926 (Univ. Sydney), Devilin Park, Kallona, *viâ* Moree.

Faulder, Kenneth Charles, M.B., Ch.M., 1926 (Univ. Sydney), 74, Bay Street, Rockdale.

Felstead, Muriel Agnes, M.B., Ch.M., 1926 (Univ. Sydney), Edinburgh Road, Castle Crag, Willoughby.

Findlay, Andrew Parkes, M.B., Ch.M., 1926 (Univ. Sydney), 38, Lang Road, Centennial Park, Sydney.

Finn, Hubert Clement, M.B., Ch.M., 1926 (Univ. Sydney), 19, St. Paul Street, Randwick.

Foley, Horace John, M.B., 1926 (Univ. Sydney), 2, Moseley Street, Strathfield.

Francis, Neil Whinney, M.B., Ch.M., 1926 (Univ. Sydney), 12, Sutherland Road, Chatswood.

Free, Edgar Grove, M.B., Ch.M., 1926 (Univ. Sydney), Aston, Maroubra Bay Road, Maroubra.

Garner, James Verner, M.B., 1926 (Univ. Sydney), St. Andrew's College, Newtown.

Gleeson, William Sydney, M.B., Ch.M., 1926 (Univ. Sydney), Rakeen, Hamilton Avenue, Coogee.

Golding, Frederick Campbell, M.B., 1926 (Univ. Sydney), St. Andrew's College, Newtown.

Goode, Caleb James Frew, M.B., Ch.M., 1926 (Univ. Sydney), Forest Road, Hurstville.

Hayes, Geoffrey Stanhope Sautelle, M.B., Ch.M., 1926 (Univ. Sydney), Kapella, Wentworth Road, Vaucluse.

Heffernan, Patrick Gabriel, M.B., Ch.M., 1926 (Univ. Sydney), Bona Vista, 48, Macalulay Road, Stanmore.

Hill, Leslie George, M.B., Ch.M., 1926 (Univ. Sydney), Beechworth Road, Pymble.

Howe, Edmund James Glanville, M.B., 1926 (Univ. Sydney), The Rectory, Gladesville.

Howe, Geoffrey Langford, M.B., Ch.M., 1926 (Univ. Sydney), The Rectory, Gladesville.

Hoskisson, Dora Ann, M.B., Ch.M., 1926 (Univ. Sydney), Springfield, Gunnedah, N.S.W.

Hudson, Mary Joyce, M. B. Ch.M., 1926 (Univ. Sydney), 161, Allison Road, Randwick.

Hull, Eric Douglas, M.B., Ch.M., 1926 (Univ. Sydney), 6, Buena Vista Avenue, Mosman.

Hungerford, Doreen Annie, M.B., Ch.M., 1926 (Univ. Sydney), Glanworth, Chandos Street, Ashfield.

Hunt, Elinor Sydney, M.B., Ch.M., 1926 (Univ. Sydney), 132, Victoria Street, Ashfield.

Jabour, Louis, M.B., Ch.M., 1926 (Univ. Sydney), Spring Street, South Grafton.

Jones, Reginald Stuart, M.B., Ch.M., 1926 (Univ. Sydney), Orara, South Grafton.

Levings, Edward Winchester, M.B., Ch.M., 1926 (Univ. Sydney), 27, Gerald Street, Marrickville.
Lilley, Alan Bruce, M.B., Ch.M., 1926 (Univ. Sydney), 15A, Roe Street, North Bondi.
Loewenthal, Louis Samuel, M.B., Ch.M., 1926 (Univ. Sydney), Rathven, St. Marks's Road, Randwick.
Lorger, Albert Eric, M.B., Ch.M., 1926 (Univ. Sydney), 109, Evans Street, Rozelle.
Lumley, George Frederick, M.B., Ch.M., 1926 (Univ. Sydney), 70, Bestic Street, Rockdale.
McEwen, Ronald James Brohan, M.B., Ch.M., 1926 (Univ. Sydney), 37, Blue's Point Road, McMahon's Point.
McKeller, Charles Crawford, M.B., Ch.M., 1926 (Univ. Sydney), 47, Gardyne Street, Waverley.
MacMahon, Edward Gerard, M.B., Ch.M., 1926 (Univ. Sydney), 77, Macleay Street, Potts Point.
MacMahon, John Stephen, M.B., Ch.M., 1926 (Univ. Sydney), 77, Macleay Street, Potts Point.
McQuiggin, Harold George, M.B., Ch.M., 1926 (Univ. Sydney), 20, Beaufort Street, Croydon.
McStay, Lionel Leslie, M.B., Ch.M., 1926 (Univ. Sydney), 250, Belmore Road, Coogee.
Mallam, Harry Roger, M.B., Ch.M., 1926 (Univ. Sydney), Highbury, Armidale.
Manion, John Allan, M.B., Ch.M., 1926 (Univ. Sydney), 134, Boulevard, Dulwich Hill.

Medical Appointments.

Dr. Harry Garnett Phippen (B.M.A) has been appointed Government Medical Officer at Junee, New South Wales.

* * * *

Dr. Leslie St. Vincent Welch (B.M.A.) has been appointed Medical Officer, Department of Public Instruction, Brisbane, Queensland.

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Dr. Kenneth Stuart Hetzel has been appointed Temporary Honorary Assistant Physician at the Adelaide Hospital, South Australia.

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Dr. Eugene Abraham Matison (B.M.A.) has been appointed Honorary Aural Surgeon (Visiting) to the "Mareeba" Babies' Hospital, South Australia.

Books Received.

BERGEY'S MANUAL OF DETERMINATIVE BACTERIOLOGY: A KEY FOR THE IDENTIFICATION OF ORGANISMS OF THE CLASS SCHIZOMYCETES, by David H. Bergey, Assisted by a Committee of the Society of American Bacteriologists with an Index by Robert S. Breed; Second Edition; 1925. Baltimore: The Williams and Wilkins Company. Royal 8vo., pp. 478. Price: $5·50 net.

THE PRACTICAL MEDICINE SERIES, COMPRISING EIGHT VOLUMES ON THE YEAR'S PROGRESS IN MEDICINE AND SURGERY: Under the General Editorial Charge of Charles L. Mix, A.M., M.D.; Volume V.: Obstetrics and Gynecology; 1925. Chicago: The Year Book Publishers. Crown 8vo., pp. 570, with illustrations.

THE PRACTICAL MEDICINE SERIES, COMPRISING EIGHT VOLUMES ON THE YEAR'S PROGRESS IN MEDICINE AND SURGERY: Under the General Editorial Charge of Charles L. Mix, A.M., M.D.; Volume VI.: General Therapeutics; 1925. Chicago: The Year Book Publishers. Crown 8vo., pp. 392. Price: $2·25 net.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xxii.

ALFRED HOSPITAL, MELBOURNE: One Surgeon to In-Patients, One Surgeon to Out-Patients; Five Clinical Assistants.
NEW SOUTH WALES GOVERNMENT RAILWAYS AND TRAMWAYS: Two Assistant Medical Officers (Male).

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C. 1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham Dispensary. Manchester United Oddfellows' Medical Institute, Elizabeth Street, Sydney. Marrickville United Friendly Societies' Dispensary. North Sydney United Friendly Societies. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association.
QUEENSLAND: Honorary Secretary B.M.A. Building, Adelaide Street, Brisbane.	Brisbane United Friendly Society Institute. Stannary Hills Hospital.
SOUTH AUSTRALIAN: Honorary Secretary, 12, North Terrace, Adelaide.	Contract Practice Appointments at Ceduna, Wudinna (Central Eyre's Peninsula), Murat Bay and other West Coast of South Australia Districts.
WESTERN AUSTRALIAN: Honorary Secretary, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Diary for the Month.

MAY 25.—New South Wales Branch, B.M.A.: Medical Politics Committee.
MAY 25.—Illawarra Medical Association, New South Wales.
MAY 26.—Victorian Branch, B.M.A.: Council.
MAY 27.—New South Wales Branch, B.M.A.: Branch.
MAY 27.—South Australian Branch, B.M.A.: Listerian Oration.
MAY 28.—South Australian Branch, B.M.A.: Branch (Annual).
MAY 28.—Queensland Branch, B.M.A.: Council.
JUNE 1.—Tasmanian Branch, B.M.A.: Council.
JUNE 2.—Victorian Branch, B.M.A.: Branch.
JUNE 2.—Western Australian Branch, B.M.A.: Council.
JUNE 3.—South Australian Branch, B.M.A.: Council.
JUNE 3.—Section of Orthopædics, New South Wales.
JUNE 4.—Queensland Branch, B.M.A.: Branch.
JUNE 8.—Tasmanian Branch, B.M.A.: Branch.
JUNE 8.—New South Wales Branch, B.M.A.: Ethics Committee.
JUNE 8.—Section of Medicine, New South Wales.
JUNE 10.—New South Wales Branch, B.M.A.: Clinical Meeting.
JUNE 11.—Queensland Branch, B.M.A.: Council.
JUNE 15.—Tasmanian Branch, B.M.A.: Council.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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